VOL. XXIII., NO. 6 APRIL, 1930.

PROCEEDINGS *of the* ROYAL SOCIETY OF MEDICINE

LONGMANS, GREEN & Co LTD
39, PATERNOSTER ROW, LONDON
NEW YORK · BOMBAY · CALCUTTA · MADRAS

Section of Comparative Medicine.

[*February* 26, 1930.]

DISCUSSION ON CANINE DISTEMPER.

Mr. Thomas Dalling: My remarks will be confined to the question of active immunization, and will deal mainly with our own experiences and those of the members of the medical and veterinary professions who have made use of the antigens made by us.

The method of immunization devised by Laidlaw and Dunkin consists in the injection of a vaccine prepared by inactivating with formalin the virus in the tissues of dogs injected with virus and destroyed at the critical point of infection, followed in seven days by the injection of a small dose of living distemper virus in the form of an emulsion of ferret spleen, the whole course being carried out under strict conditions of isolation of the subject, &c. In a series of over 2,000 dogs, these workers showed that this method was effective in at least 99% of cases.

It was at this time that we were asked to undertake the making and issue of the prophylactics on a large scale, to members of the medical and veterinary professions. Our experiences, however, cover the use of the antigens on many thousands of dogs all over the country, and refer to vaccine and virus used on a commercial scale more than on an experimental scale.

They confirm those of Laidlaw and Dunkin in that the antigens devised by them do produce an active immunity to natural and artificial infection with distemper virus, and that, provided potent vaccine and virus are used, the method is successful to a very high degree.

On the whole, the results of the use of the prophylactics appear to have been good. Naturally, we did not expect that 100% efficiency would be recorded from their use, and we expected to hear of failures. It is with these failures that we are chiefly concerned now. That some veterinary surgeons have apparently had nothing but excellent results is evident from some of the reports received. For example, a veterinary surgeon who had used twenty-eight doses at various times, says that all his cases have been absolutely without incident, and places a high value on the efficacy of the vaccine. Another writes that after using 140 doses, he has had 100% success, and that he has inoculated three kennels of foxhounds, and in each case the Master of the Hunt has seen fit to speak in public on the efficacy of the vaccine. Again, after the use of 217 doses the report is that it is 100% successful. Recording good results from the use of eighty-seven doses, a member of the profession says that his experiences are that it gives satisfactory results in more than 99% of cases, and that if care is taken that the dogs are in good health at the time of inoculation, the method is 100% successful and free from risk. These are a few of the good results culled from the reports received.

There have been, however, a number of untoward happenings which may conveniently be divided into three groups: (*a*) illness following immediately after the injection of vaccine or virus, or both, (*b*) nervous disturbances following the use of the prophylactics immediately or months later, (*c*) breakdown in immunity a considerable time after the use of the prophylactics.

Illness following immediately after the injection of vaccine or virus, or both.—Our experience at the laboratory and that of probably 95% of veterinary surgeons is that the administration of vaccine is followed by little or no departure from normal health, provided that the dogs at the time of receiving the vaccine are healthy and have not been recently exposed to infection of any kind. Occasionally an unpleasant report of illness after vaccine administration is received. For example, we have a record of a whole kennel of eight dogs suffering unduly fourteen days after the injection of vaccine. Without virus having been given, all the dogs showed what the veterinary

surgeon described as "definite symptoms of distemper," and five died. Our records show, however, that illnesses following the use of vaccine are very rare indeed. When virus is injected seven or more days after vaccine, in a proportion of cases there is evidence of a disturbance of the general health of the subject in that, within a week, there may be a rise in temperature, lack of appetite, watery discharges from the eyes and nose and a slight diarrhœa. As a rule these symptoms pass off and the dog soon returns to normal health. In some subjects these symptoms may be exaggerated, and the dog may be severely affected, and is only by careful nursing restored to health, while in other cases actual death takes place within ten or fourteen days after the use of virus. It is exceptional to find an exaggeration of the more or less slight disturbance from normal health, but it does occur, and in their original instructions issued with vaccine and virus Laidlaw and Dunkin state that "experience shows that the risk of death from double vaccination is less than 1%." The reports of illness and death following the injection of virus may refer to one individual dog in a kennel in which any number up to twenty or thirty are being immunized at the same time, or on the other hand it may refer to almost every dog in the kennel which received the virus at the same time. Reports of the former nature call for little comment except that the individual concerned was in some way more susceptible to the action of the virus than his fellows. In the latter case, however, a more serious view must be taken, and we have, as far as possible, investigated the mishap and tried to come to some conclusion as to its cause.

Nervous disturbances following the use of the prophylactics immediately, or months later.—Attributed to the effects of vaccine and virus are fits of all degrees and variety. It has been assumed by some veterinary surgeons that hysteria has been actually caused by vaccine or virus, or that the injection of these materials has in some way predisposed to the development of hysteria. Symptoms of hysteria have in some cases been noted on the day following the use of vaccine; in other cases they have appeared within a week of the use of virus, while in further cases their appearance has been noted weeks or months later. The same remarks apply to fits usually described as "distemper fits." In rare cases chorea symptoms have been noted, following the milder evidence of distemper in immunized dogs, and in one case a veterinary surgeon describes a true encephalitis, confirmed by examination of sections of brain. The suggestion is that these conditions are either directly or indirectly related to the use of vaccine and virus. It has also been suggested in the case of fits developing months after the course of immunization that their occurrence is evidence of a breakdown in immunity, the assumption being that the occurrence of fits is more or less diagnostic of a distemper infection.

Breakdown in immunity a considerable time after the use of the prophylactics.—A number of reports have been received describing diseases in immunized dogs, which the owners and the veterinary surgeon have called distemper; in other words, there has been in these cases an apparent breakdown in the immunity produced by the injection of the vaccine and virus. We have been specially interested in these reports and have, wherever practical, visited the sick animals, or received material, such as swabs of nasal and eye discharges, spleens, etc. The alleged breakdowns refer to single dogs owned by private individuals as well as to groups of dogs in kennels, especially hunt kennels, the hounds of which were inoculated as puppies at walk and became sick when introduced into the kennels at a later period. Most of the research work we have carried out has been on material received from such cases. We have a considerable amount of evidence to show that the active immunity described by Laidlaw and Dunkin is borne out by field results. For example, the following are typical of many reports we have had. A veterinary surgeon, describing his results of immunizing forty foxhounds, says that after introduction to the kennels they have been in contact with dogs affected with distemper and none have contracted the disease. Another veterinary surgeon says

that he definitely exposed two vaccinated hound puppies to distemper infection and no signs of illness were observed in either pup. Writing on the results of inoculating twenty dogs, a member of the profession states that all the dogs have gone on remarkably well notwithstanding that the kennels in which they are now housed are hotbeds of distemper. A further example is given by a veterinary surgeon who, in February last year, inoculated twenty foxhounds, which all resisted distemper when it broke out in the kennels later in the spring. As opposed to these good results, the following examples may be quoted as typical of the alleged breakdowns. Two retriever pups were inoculated by a veterinary surgeon in July; in September both pups were reported as having contracted "distemper" and one died. Thirty-four couples of foxhound puppies were inoculated at walk by a veterinary surgeon; six months later they were brought to the kennels, in which distemper was present. Sixteen of the puppies developed distemper and two died. Another case relates to an outbreak of distemper, described as a "perfect picture of distemper" among seventy-eight foxhound puppies, inoculated at walk and returned to the kennels some months later. These examples could be multiplied, but these will serve as an indication of the results experienced.

Some possible explanations of mishaps: (1) *Quality of vaccine and virus.*—The important point in the consideration of mishaps or failures is the quality of the vaccine and virus used. Laidlaw and Dunkin laid down a method of testing vaccine and, as far as possible under conditions attending the making and testing of large amounts, we have adhered to their method, and have only issued vaccine which has, in our hands, fulfilled the requirements of the test. It can readily be understood that this has meant the discarding of a proportion of the vaccine made, as we have not been able to show that all the batches made were up to the standard laid down. These of course were not issued. In the making and testing of batches of vaccine, many important points have to be learned by experience, e.g., the exact time at which to obtain the tissues so that they will contain the maximum amount of virus, etc. We feel, however, that we are now acquainted with the various pitfalls in the making and testing of vaccine, so that the task is not now so arduous. A further difficulty we have experienced is in preserving the living virus so that it will reach the injector's hands in a fully potent condition. It is essential to the Laidlaw-Dunkin method that the injection of one dose of vaccine should be followed by the use of living virus, in order to obtain the greatest degree of immunity. We are not satisfied, even now, that the method we have adopted in the issuing of the living virus is the best conceivable, and we are still carrying out experiments to discover if the present method of issuing virus can be improved in order that it may be more fully potent when used as a second injection.

(2) *Condition of the dog and exposure to infection.*—A possible explanation of some of the mishaps, especially those relating to the occurrence of so-called distemper or exaggerated reactions after vaccine and virus, is the inoculation of dogs which have been recently exposed to infection, especially with naturally occurring distemper virus, and are, in all probability, in the incubative stages. Great care has been taken in the formulating of instructions for the guidance of the immunizer and particular stress is laid on the recording of temperatures before immunization is begun and during the whole course. An elevated temperature must be taken as some guide to infection with naturally occurring distemper virus or other organisms and serves as an indication to withhold the administration of vaccine or virus. A considerable amount of evidence has been accumulated on this point and shows that many mishaps soon after the administration of virus have been due to a disregard of this important instruction. The careful veterinary surgeon who insists on an accurate record of the temperatures being kept before the dogs are subjected to the treatment, usually escapes any untoward results following the use of vaccine and virus. One veterinary surgeon says he insists that the owner should

take the temperatures twice a day before the first injection, and in several cases in which he had doubts of this having been done, he declined to use the vaccine. We have also records showing that dogs have been definitely exposed to distemper infection during their course of vaccination. An example of this occurred in the practice of a veterinary surgeon who discovered, after he had inoculated several couples of foxhounds in the kennels, that they were on a run in which dogs had recently died from distemper infection. The method is perfectly safe as far as setting up of diseases in dogs is concerned, provided the dogs are suitable subjects for inoculation, and that the instructions laid down are carried out. The use of a living virus calls for the strictest attention to detail, and more care is required in its use than in the use of an ordinary dead or killed vaccine.

(3) *Age of treated dogs.*—It is recommended by the originators of the method that puppies should be three or four months old before immunization is carried out and this is the advice we give when consulted on the point. We have records of dogs being injected at all ages, from six weeks upwards, with varying results. Personally, we are of opinion that while puppies are still suckling, the possibility of maternal immunity transmitted in the milk must not be overlooked. It seems possible that in some cases a sufficient number of antibodies will be transmitted to vitiate the antigenic action of the injected vaccine and virus. The usual history of the results of the inoculation of such young puppies is that they showed no reaction whatever to the vaccine or virus. It is difficult to predict how these puppies will behave when exposed to natural infection at a later date. We are now carrying out experiments to obtain evidence on this point. It would appear also from the experiences of other veterinary surgeons that dogs older than three or four months suffer more reaction from virus than do younger dogs.

(4) *Wrong diagnosis.*—We have carried out post-mortem examinations on a large number of dogs which have died either as a sequel to the use of vaccine and virus, or to an attack of so-called distemper some time after inoculation, and, in addition, have examined a considerable amount of material received, in the form of nasal discharges, etc., on swabs. Laidlaw and Dunkin have shown conclusively that the occurrence of the distemper virus in a tissue can, under laboratory conditions, be detected by the use of the highly susceptible ferret. In alleged breakdowns it is therefore a fairly simple matter to prove whether or not the distemper virus is present. Spleen tissue, blood taken from the dog while the temperature is high, or nasal discharges in the initial stages of the infection in an infected dog, contain an abundant supply of distemper virus. The behaviour of a ferret injected with any of these materials should prove definitely whether or not the virus was present in the dog. The sum total of our work up to date is that in a small percentage of the cases examined we are satisfied that the distemper virus existed in the tissues of the immunized dog examined by us, and in these it must be considered that the immunity produced was insufficient to protect against natural infection. I understand from Dr. Laidlaw and Mr. Dunkin that they have also been able to show the presence of the virus in the tissues of some immunized dogs which have later suffered from distemper owing to a "breakdown of immunity." In some cases of apparent breakdown in which we have failed to demonstrate the presence of the distemper virus, the symptoms shown were closely allied to those produced in susceptible dogs by injection with distemper virus. From our investigation work we conclude that symptoms of disease which may be confused with those produced by the virus of distemper, may be produced by causes other than the distemper virus. In confirmation of this I may refer to an experience at the laboratory. An epidemic occurred among some dogs in isolation, previous to their being used for the production and testing of distemper vaccine. It was characterized by nasal and eye discharges, coughs, diarrhœa, high temperature, etc., and several dogs died The opinion of several veterinary surgeons was that the disease was distemper.

The post-mortem examinations showed the presence of pneumonia in various stages. A careful search was made for the distemper virus in the discharges of the living dogs, in the blood of the living dogs, and in the spleens of dogs specially killed in all stages of the disease. The results were entirely negative, but a hæmolytic streptococcus was isolated in pure culture from the lungs of most of the dogs. It is worthy of note that a hæmolytic streptococcus has been isolated, mostly in pure culture, from the lungs of immunized dogs which have subsequently developed so-called distemper. In some cases the virus was also demonstrated in the tissues.

The general opinion in the past has been that the virus of distemper is the primary cause of all diseases classified as distemper, and that other conditions affecting dogs are due to a secondary cause acting in the presence of the virus of distemper. Without doubt, in the field, true virus disease does exist, but more commonly secondary organisms invade the tissues and give rise to lesions, etc., which may cause the death of the animal. In addition, however, in our opinion some, at least, of these so-called secondary conditions may exist as primary, without the presence, at any stage, of the distemper virus. It is known, for example, that *Bacillus bronchisepticus* may infect dogs and produce evidence of clinical distemper without the association of the distemper virus. We, ourselves, have been able to infect dogs proved to be highly immune against the distemper virus, with cultures of this organism, and it was the opinion of several veterinary surgeons who saw the cases that the symptoms set up were those of canine distemper. At the present moment it would appear that *Bacillus bronchisepticus* is rare in this country, but some years ago it was the simplest matter to isolate this organism from dogs affected clinically with distemper. It is possible that such a disease as bronchisepticus infection may appear as a wave of infection.

From these and other observations, I am of the opinion that we should not assume that all cases of alleged failures after immunization should be classified as distemper. Without doubt some are true failures, in that the virus can be demonstrated in the tissues, but there remains the group in which the virus cannot be found in dogs in any stage of the disease but in which organisms in pure culture can be isolated. A further study of such conditions is indicated, and the valuable ferret test devised by Laidlaw and Dunkin is of the utmost assistance in guiding one as to the cause of any outbreak simulating distemper.

Mr. George W. Dunkin: The title of this discussion was at first, "The Present Position of Distemper Immunity," but more recently it has been called "Canine Distemper." It is, however, the subject of immunity which should be discussed, owing to the publicity given to it, particularly in the lay press. In my own view, the position to-day is precisely the same as it was a year ago. That is to say, it is possible to confer an active and lasting immunity on dogs and ferrets by means of suitable agents. Just over a year ago my colleague, Dr. P. P. Laidlaw, and myself published our findings, based not only on a considerable number of laboratory experiments carried out with meticulous care and under almost ideal conditions, but also on a large field experience covering some 2,000 dogs and hounds scattered over various parts of the United Kingdom and treated by over ninety veterinary surgeons. A complete record was obtained of these cases and it was found on analysis that deaths from all causes following on the inoculations amounted to less than one per cent. More recently, as has been stated by Mr. Dalling on several occasions, the work of manufacturing and distributing the products has been undertaken commercially and many thousands of doses of vaccine and virus have been distributed.

It has been said on more than one occasion that the clinical symptoms of distemper as seen in the field and those which have been described as experimental distemper, have certain differences. It is not surprising that differences of this nature should occur, for on the one hand we have a disease occurring in perfectly

natural conditions and in places and surroundings which might invite complications; on the other hand, the disease is carefully controlled and the symptoms seen are those of the simple type uncomplicated by other pathogenic organisms. In our experience the most common secondary invader is a streptococcus, and pneumonia is commonly seen in cases where this organism is found. I believe I am correct when I say that in all our experimental work we have only seen one case of pneumonia.

It is a well-known fact that pneumonia is a common complication of ordinary field distemper, and deaths are usually brought about in this way except in those cases which show evidence of cerebral infection. Even in these pneumonia may be present.

In view of this admitted difference in the post-mortem findings of field and experimental distemper, it was thought by some that we had not been dealing with that form of distemper which the clinician is called upon to treat in his daily work. It is true that the majority of dogs which have recovered from a natural attack of distemper appear to be immune to further attacks, even though cases with which they are subsequently in contact are the subjects of streptococcic and other bacterial infections, in addition to the virus of distemper. Practical experience has shown that vaccination with any of the many bacterial vaccines on the market will not protect against distemper, for although they have been in use many years, the disease continues its sway and takes its usual toll. The question therefore which remains to be settled is: "Will dogs which have been immunized against distemper by the vaccine-virus method also be protected against the bacterial infections which are seen in the field?" It was largely for this reason that a certain amount of field work was undertaken after our laboratory experiments had given us some encouragement, but it is only recently, as one might expect, that evidence on this point has been forthcoming. I have been privileged recently in visiting five large foxhound kennels in such widely separated districts as Scotland, Yorkshire, Bedfordshire, Wiltshire and Devon. In all these kennels young hounds were going down with distemper. It is agreed by all veterinarians that the absolute and certain diagnosis of distemper as it occurs in the field is not an easy matter and although the cases which I saw in these kennels strongly supported the view expressed by the Masters, their kennel huntsmen, and the visiting veterinary surgeons, it remained to be proved beyond cavil that such was the case. In many instances we have succeeded in proving the presence of the virus of distemper by the injection of emulsions of spleen from the carcases of dead hounds. It is fair to say that quite 99% of all the hounds which have been examined have had pneumonia, so that there was a double infection at least. In all these kennels there were also older hounds which had either recovered from naturally occurring attacks or had been immunized over a year ago. There appears to have been no case of breakdown among these hounds and the opportunity of contracting infection was present daily. It is not possible in most kennels of foxhounds to isolate sick from healthy hounds, for even in those kennels where a hospital has been provided, indirect infection is probable and segregation of attendants is not practicable.

As a result of my series of visits I am satisfied that a dog or hound which has been protected by the injection of a vaccine of good quality, followed by living virus, is not only protected against distemper, but against the usual secondary infections met with in such cases.

The question of breakdown in immunity has to be referred to. As far as our own experiences are concerned, I have said that deaths from all causes amounted to less than 1%, and this applies to the series which we reported in November, 1928. Since then we have had one other case—a Japanese spaniel—which developed illness and subsequently died. In life this little animal showed all the signs of distemper and we succeeded in obtaining proof of the presence of the virus of distemper from the blood and the eye washings. This case must be added to the

list of breakdowns. Reputed breakdowns have occurred on a somewhat larger scale since the production of the vaccine and virus was placed on a commercial basis, and it is necessary that one should not come hastily to a conclusion as to the cause. In referring to these reputed breakdowns one must make reference at this stage to the clinical symptoms of distemper. I have already stated that the certain diagnosis of this condition is by no means easy, and I am quite sure that the experienced canine surgeon will be the last to hold dogmatic views on such a debatable point. Apart from nervous affections simulating distemper, we have to differentiate it from *Bacillus bronchisepticus* and streptococcic infection, and this cannot be done with certainty without the aid of the laboratory.

The preparation of the agents under discussion is one which requires infinite care. The patients must be carefully selected at the proper time. The tissues must be collected under strict aseptic conditions and carefully tested for sterility before issue. The vaccine must be tested for protective properties on dogs known to be susceptible, for it is not uncommon to prepare a vaccine with all the stated precautions only to find on test that it is useless. The virus, being a suspension of distemper ferret-spleen, must be collected from ferrets which are suffering from distemper and distemper only, and its potency must be known. Finally, neither product must be used on dogs which are not in a perfect state of health, and a careful temperature record both during and before vaccination should be made. The preparation of these agents on a commercial scale must, I should imagine, be a much more difficult matter than its manufacture on a smaller laboratory scale, and doubtless the difficulties are great. It is necessary, for example, to have on hand a supply of vaccine whenever it is demanded; this applies to virus also, and, since the virus of distemper is unstable, I can see at least some of the difficulties with which the manufacturers have to contend. Fortunately it is none of my business to clear up these difficulties, and I only make reference to those which I believe may be the more serious ones, because it is necessary to ascertain exactly the cause of the recent breakdowns. It is not possible at the moment to say definitely what the cause is, but doubtless those who have the manufacture of the products in hand will decide this point. One thing is clear, and that is that a state of immunity which is active and durable can be induced in susceptible dogs by means of the process referred to. I do not believe that a lasting immunity can be conferred by the injection of vaccine without living virus, at least not by one injection. Whether multiple doses of vaccine will bring about this desirable state of affairs no one can yet say, for to prove such a point it would be necessary to vaccinate a group of dogs known to be susceptible, to keep them absolutely isolated and, after varying periods extending to several years, test them for immunity with virus of a known virulence.

Mr. Dalling has referred to certain complaints which have come to his notice, and I should like to comment on one of them, namely, "illness following immediately after the injection of vaccine." It does not seem to have been sufficiently understood that the vaccine, properly prepared, cannot give rise to any illness. It consists of a 20% suspension of finely minced dog-tissue containing originally living distemper virus which has been killed by the action of a certain amount of formalin. It has been observed that the presence of the formalin appeared to induce certain irritant effects after injection, and therefore a small amount of ammonia is now added, sufficient to bring the pH up to about eight, thus converting all free formaldehyde into urotropine in order to avoid this undesirable effect.

Illness immediately following the injection of vaccine must, therefore, have a cause other than the mere introduction of this material (I do not refer to the incidence of local abscess but to systemic disturbances). In the event of a dog developing symptoms of distemper at this stage of the process before virus is given, there is, in my opinion, only one explanation, and that is that the patient was in the

incubative stages of the disease when he received the vaccine. It was for this reason that in certain cases when the procedure was practicable, the taking of bi-daily temperatures for about a week before vaccination was advocated. In the case of individual and possibly valuable animals this can be done. The possession of such a record by the veterinarian provides an invaluable guide as to whether he shall or shall not proceed with the vaccination process. Severe illness immediately following the introduction of virus is due to one of two causes—distemper or some other infection. If the latter, then clearly neither vaccine nor virus can be held to blame, for on the one hand we have material which consists of dead virus suspended in homologous tissue, and on the other, living distemper virus suspended in finely minced ferret spleen. If the illness is distemper then the cause is due either to the hypersensitiveness of the patient, or to the fact that insufficient protection has been afforded by the vaccine previously injected.

Dr. S. G. Billington said that he had been investigating canine distemper for seven years. The objectives had been: (1) To isolate the causal organism. (2) To evolve a curative remedy. (3) To consider the question of prevention.

With regard to the causal organism, he had come to the conclusion that it was the filter-passing stage of the *Bacillus bronchisepticus*, and for six years he had had the three stages of filter passer, bacillus, and intermediate leptothrix, growing and reproducing as such, or being converted into the other stages at will.

With regard to the curative remedy: in the practical consideration of any curative product, there were obviously three clinical groups of distemper: (i) The very early, pre-secondarily infected cases. (ii) The pre-moribund cases, but with superadded secondary infections. (iii) The moribund cases. Group iii could be dismissed, and the great majority of the remainder as met with by the practising clinician would be in group ii.

For several years curative vaccines for the three stages had been used and the results critically watched. Vaccines of filter passer and leptothrix consistently controlled the early, uncomplicated disease, but in cases with superadded secondary infections the results were variable and uncertain. As, however, this group was much the largest one met with in practice, it was the most important one, and there appeared to be two methods of approach: (1) To produce a blunderbuss vaccine containing the common complicating organisms, or preferably (2) To isolate and extract that something known as "natural immunity" as existing in the serum of the naturally immune mongrel.[1]

The product termed curative distemper inoculin was virtually "concentrated natural immunity," and distemper in the pre-moribund stages was controlled in an extremely high percentage.

With regard to a preventative—most preventative vaccinations were effective for a period only. With a disease like distemper, which was always with the canine community, to be of practical utility it must be permanent.

He (Dr. Billington) had decided to await the results in susceptible dogs which had survived distemper owing to the help of an anti-distemper remedy. The following results were typical of a number observed: Four years ago, a pack of foxhounds had developed distemper. With the use of a curative vaccine the mortality was *nil*. In the usual spring outbreak a year later, about 20% of the dogs cured the year before, contracted the disease, probably representing about half the number that would have died without the help of the curative vaccine. One could not hope to get any preventative vaccination more potent than an attack of the disease, and, consequently, no attempt to produce a preventative had been made. He thought that the weakness was an endocrine one, resulting in failure to retain the acquired immunity.

[1] *Medical World*, 1929, xxix, 310.

Mr. A. A. Comerford said that during the three years in which he had used this treatment he had obtained favourable results until recently, and he would like to refer to three packs of foxhounds treated in August, 1929. One pack had, so far, shown no bad results. In another, early in 1930, distemper had been reported, though personally he was inclined to doubt its presence. The affected hounds ran temperatures up to 104°, and had discharges from eyes and nose, but never lost their appetites or their spirits, and though a few casualties occurred, a ferret survived all attempts at infection. The third pack undoubtedly had a definite outbreak of distemper with all the clinical symptoms, and a ferret which was allowed to run about the kennel for ten minutes every day while the sick hounds were out in the yard, showed clinical distemper symptoms on the sixteenth day. This was undoubtedly a breakdown, and he (the speaker) would like to ask Mr. Dalling one question: Was he quite confident that the virus (used within the time limit specified on the label attached to each vial) was potent? One reason why he was inclined to doubt that conclusion was suggested by an experience last summer when he was immunizing a litter of puppies eight weeks old. Through a mistake on the kennelman's part, another litter was put into the kennel containing the puppies which had just received their dose of virus. No bad results followed amongst the non-vaccinated puppies, though there was every reason to believe that those which had received the virus should have been infective if the virus had been potent. With reference to the duration of immunity, he, personally, could vouch for foxhounds immunized three years previously who had stood up against several outbreaks without any symptoms developing, and he had no hesitation in stating that in his opinion that immunity was for life.

Dr. R. A. O'Brien said he was deeply interested in the point Mr. Comerford raised. Virus was unstable. It was much easier to be perfectly sure that the virus reached the injector fully potent and alive when, as during the experimental period when Dr. Laidlaw and Mr. Dunkin were sending out virus, they kept the material frozen hard until the last moment and dispatched it when they chose. Mr. Dalling did this also during the experimental issue of 2,000 doses at the commencement of his work. But later when it became necessary to meet a large demand and to have the virus constantly available, the difficulties became great, and it was quite possible, as Mr. Comerford suggested, that in some few instances the virus was not fully potent when it reached the dog. Mr. Dalling was again investigating this point. By keeping the material frozen hard until the last moment and by shortening the period during which a batch of virus might be used, it would probably be possible to continue safely the issue of the liquid virus. If not, it could probably be issued dry after full tests; there was still another method being tried. The articles in the daily press had been mentioned. It was wise to preserve a sense of proportion and of fairness. The inquiry sent to veterinary surgeons recently had yielded reports relating to about 12,000 injections. The total complaints, mostly of alleged failures of immunity at a later date—even if we debited every one against the prophylactic—amounted to 8% and the total deaths to 2%.

Mr. P. P. Laidlaw said that the reputed breakdowns in immunity formed, for him, the most interesting aspect of the discussion. He believed that in the great majority of cases a dog which had survived distemper was immune for life, and, indeed, the justification for the injection of living virus, as a final step in the immunization process, was based on this conception. Distemper which appeared in an animal which had once received living virus was thus, for him, of the greatest interest. The diagnosis of distemper was often difficult and the differentiation of pneumonia from distemper complicated by pneumonia seemed almost impossible. It was sometimes possible, however, to secure proof of a distemper attack by the

transference of a typical disease to a susceptible animal—such as a ferret—utilizing material from a sick dog. When the result of such an inoculation was positive, there could be no doubt as to the interpretation of the experiment, but when such tests were negative, there still remained the possibility that distemper virus had been present in the dog at some earlier stage of his illness. He was of the opinion that a better chance of transmitting the virus occurred in the early than in the late stages of the disease, and that in the case of post-mortem material the chances were not good. However that might be, it seemed to him that the number of cases of proved lapses in immunity was greater than might be expected from the recognized immunity of a dog which had survived distemper. It was essential to study all cases of failure with the greatest care, in order to be certain that the failure was a genuine lapse in immunity, and then, by improvement of technique, to try to reduce such failures to the lowest possible level.

There was no evidence—as yet—of distinct strains of distemper virus, such as had been shown to exist in foot-and-mouth disease. All strains so far studied (including one American strain) cross-immunized. A dog or ferret which was immune to a strain from one source appeared to be immune to strains from other sources.

He (the speaker) was at variance with Dr. Billington's belief that a dog might have multiple attacks of distemper, just as he was completely at variance with Dr. Billington's findings regarding blood-cultures from distemper cases. In the publications of Dr. Billington which he had read, he had not found precise directions as to the methods by which *B. bronchisepticus* might be transformed into "the filter passer" or alternatively into "the streptothrix," and thus it had not proved possible for him to take stock laboratory culture of *B. bronchisepticus* and test Dr. Billington's views on this aspect of the subject.

Mr. Dalling (in reply) said that the most important point of the discussion had been raised by Mr. Comerford, namely, the question of the viability of the virus when it had reached the injector's hands. There had undoubtedly been some "breakdowns in immunity," as shown by the recovery of the virus from the tissues of dogs which had been inoculated with vaccine and virus, and had later shown clinical evidence of distemper. This could be due either to the injection of vaccine of poor antigenic value, or to the use of non-living or non-potent virus. He was inclined to think that poorly antigenic vaccine had not been used, because of the rigid tests employed before the issue of any batch: on the other hand, the virus issued might have been at fault in a small number of cases. It had been found most difficult to issue virus of a constant potency. An examination of a sample of virus issued for use had shown that that special ampoule had contained virus which would not infect a ferret. Because of the records of reactions noted by veterinary surgeons, it was evident that the huge bulk of the virus issued was potent, but it seemed probable that some had fallen short of the required standard to consolidate the immunity produced by the vaccine. New methods were being investigated in an attempt to improve the issue of virus.

Section of Medicine.

[*February* 25, 1930.]

DISCUSSION ON THE TREATMENT OF LUNG ABSCESS.

Dr. F. G. Chandler: In a discussion on the treatment of abscess of the lung, ætiology and diagnosis are not germane. I must, however, make two transgressions: (1) when the cause affects the treatment, (2) when localization influences the route of approach.

It is essential to exclude a foreign body as the causatory factor. A tooth, a tooth-stopping, a fruit seed, a small piece of bone, and many other things may be aspirated. A careful history, therefore, is essential, also an X-ray examination, and in certain cases bronchoscopy. Bronchoscopy should be employed if there is the least doubt, as the removal of the cause may cure the abscess.

Localization will be made by the physical signs, which, however, have strict limitations, and by X-ray examination. One of the most valuable guides is the true lateral skiagram. Stereoscopy is of doubtful value.

If no foreign body is present, medical treatment should first be adopted. Firstly, because Nature frequently asserts herself and the patient coughs up the abscess with complete recovery. Secondly, because, given time, the abscess, if it does not disappear, may localize itself into a definite, rounded, walled-off cavity, thus facilitating surgical treatment and making it most hopeful.

The medical treatment at our disposal is as follows: Rest in bed, attempting postural drainage from time to time; poultices; inhalations, such as steam inhalations with friar's balsam, and menthol: inhalations of carbolic, creosote, chloroform, iodine or ether drops on a Yeo's mask; opium and its derivatives, hydrocyanic acid and allonal, etc., in order to suppress excessive coughing; and treatment of oral and nasal sepsis, if present. This last is all-important. Some cases of abscess are, I believe, directly due to oral sepsis. Seeing that a spirillum is not uncommonly present in the expectoration from an abscess, arsenic in its various forms is often employed; certainly no harm is done by using this drug, and it may do good. Medical diathermy I should put in the same category; it is not an unreasonable treatment.

The question will naturally arise, "How long should we persist in medical treatment?" The answer depends on many factors, such as the urgency of the symptoms, the tendency of the abscess to localize itself, and whether the necrotic material has reasonably free escape through the bronchus. It is largely a matter of judgment and of the individual peculiarities of the case. Dangerous coughing attacks would necessitate operation, as would deterioration of the patient's condition, or failure to improve, X-ray evidence of the abscess becoming larger, or evidence of continued toxic absorption. We must also consider the possibility of subsequent fibrosis or bronchiectasis.

Lipiodol injections.—Lipiodol is often injected in these cases for the purpose of diagnosis: chiefly because it is essential to know whether there is an abscess or bronchiectasis, or both. I have made this injection in a number of cases of abscess, and I have thought that it helped towards spontaneous recovery. In some cases I have injected lipiodol two or three times. It is difficult to be certain of its efficacy, as spontaneous recovery is common, and if lipiodol helps it may be because of its lubricant action. It does no harm; given gently it distresses the patient very little, and frequently not at all. It is invaluable in differential diagnosis, and will have to be given in a proportion of cases for this reason. The following is a striking example of the apparent efficacy of lipiodol:—A patient, aged 54—a Jewess—had both diabetes and a lung abscess. She was gravely ill, and the physical signs suggested that the abscess might be secondary to a malignant growth. There was high fever,

abundant foul expectoration and dullness over the whole of the left upper lobe, which was completely opaque to the X-rays. I injected 25 c.c. of lipiodol as a diagnostic measure. After the injection the patient rapidly became well, and the whole of the lobe cleared up completely. Injections of other antiseptic oils have been tried, such as gomenolized olive oil, but we are less certain of their value than we are of lipiodol, and they do not help in diagnosis. I have wondered whether argyrol drops given by the oral route would help, but have not tried them.

Bronchoscopy.—I have already referred to the diagnostic value of bronchoscopy. Some would claim for it a place of importance in treatment. I am inclined to be sceptical of its value, except when a foreign body is present and can be removed, or when there is some other removable obstruction of a bronchus, or in the rare case in which a stricture of a bronchus may be overcome by dilatation or permanent intubation. A case of abscess of the lung distal to a syphilitic bronchial stenosis under my care at the City of London Chest Hospital, was so treated by Mr. Howarth. The patient had periodic attacks of fever, malaise, cough and foul expectoration. He was first treated by artificial pneumothorax and the usual anti-syphilitic measures, but these did not cure him. The intubation appeared to establish better drainage. The patient was discharged from hospital with the tube in, in order that we might test its efficacy. Unfortunately he died a few months later from bronchopneumonia. But I do not think that either the intubation or the anti-syphilitic treatment was effecting a cure. Hypothetically it might be argued that by passing a probe or catheter through the œdematous-walled bronchus, better drainage might be promoted, and so help spontaneous cure, or the localization of the abscess; but it is just this that one looks for from medical treatment, and the bronchoscopy would have to be done just when we should least want to disturb the patient.

Artificial pneumothorax.—This treatment would be simple and attractive if it acted. Unfortunately the dramatic cures we may achieve in pulmonary tuberculosis are not, as a rule, seen in cases of lung abscess. There are several fairly obvious reasons for this, such as a danger of enclosing necrotic material, especially when there is not free drainage of the abscess into a bronchus. Above all, there is a danger of an empyema forming. This is a serious complication, because it may entirely prevent the adequate surgical treatment of the abscess subsequently, as it renders both localization and approach exceedingly difficult. If the abscess is superficial there is a special danger of empyema; if it is deep-seated there is a danger of enclosing pus or necrotic material, unless, as I have said, there is free drainage into a bronchus, in which case we may hope for a spontaneous cure. If, however, this spontaneous cure does not take place, then possibly artificial pneumothorax treatment may be indicated. I, personally, would prefer other methods of treatment. With a diffuse uncircumscribed abscess I think that artificial pneumothorax treatment may be worth trying, but, so far as I know, the experience has not been very good. The case described as a brilliant success in Lillienthal's work on thoracic surgery might illustrate equally well a spontaneous recovery.

Operative treatment.—Treatment will depend to some extent on the type of abscess. With a definitely localized encapsuled abscess which is not healing under the treatment mentioned above, drainage is the best treatment. With the uncircumscribed abscess or multiple abscesses, or that condition in which it is uncertain whether one is dealing with abscess or bronchiectasis, frequently all that can be done is to cut into the lung and put in a drainage tube. In some cases, however, partial lobectomy may be performed, preferably by some instrument that minimizes bleeding. For this purpose the actual cautery and the steam cautery of Souttar have been used, and, latterly, the diathermy cutting wire, which is probably the most satisfactory method. In some cases of chronic abscess, even complete lobectomy might be indicated, but one is unlikely to find the affected lobe not bound

down in places to the parietal pleura by adhesions, and a successful lobectomy depends to a large extent on the absence of dense adhesions.

In draining an abscess it is essential to have the two layers of the pleura adherent at the place where the incision of the lung is to be made. If not, there is almost a certainty of an empyema, and when the pleura is opened the lung will fall away, making the operation difficult, and adding to the risk of aspiration into the other lung, and no stitching of the pleural layers together is, to my mind, satisfactory. The rib should be resected subperiosteally, with no damage whatsoever to the parietal pleura; even a small nick may be disastrous. It must then be determined whether the two layers of the pleura are adherent or not. They may be seen moving upon each other, or it may be possible to lift up the parietal pleura by dissecting forceps. If there is the least doubt, it should be assumed that the layers are not adherent, the outside of the parietal pleura should be painted with iodine and the wound packed with iodine gauze, and left for from four to six days. After this time the two layers will be adherent and the second stage of the operation can be performed.

Sometimes a pleural effusion forms over the region of the abscess. This may create considerable difficulty. I had such a case recently and replaced the fluid in order to secure a skiagram that would show the abscess. I regretted doing this afterwards as the air did not absorb properly and it was found impossible to make the pleural layers adherent by the method I have described. Another time I should attempt to withdraw the effusion by several aspirations, if necessary, and then have the skiagram taken. In such a case if it is impossible to make the pleural layers adhere, it would be justifiable to stitch the lung to the chest wall. If an empyema has already formed over an abscess, that again makes the treatment very difficult. It might be advisable first to replace the empyema, then make an X-ray examination to localize the abscess, if possible, and perhaps keep up the pneumothorax for a few days in order to see if this diminished the expectoration. The empyema would then be drained in the ordinary way and if the treatment of the abscess was urgent it would be necessary to open the lung into the already infected pleural cavity. The difficulty, of course, would be in the accurate localization of the abscess. If a broncho-pleural fistula had already formed, there would probably be no need for any incision of the lung.

Sometimes, after successful drainage of the abscess, a sinus persists. The walls of the cavity will not approximate. This is the indication for phrenic evulsion which is a most successful procedure. Sometimes it is employed as a primary measure. Morriston Davies quotes a successful case in which it was carried out as a preliminary to thoracoplasty.

The following case illustrates nearly every point in the treatment of lung abscess.

A boy, aged 12, was sent to me in 1925 coughing up large quantities of purulent foul sputum, with occasional hæmoptysis and night sweats. The fingers were clubbed. He was wasted. There were abnormal signs at the right base. A diagnosis of bronchiectasis had been made and the case was regarded as hopeless. Lipiodol was injected and would not flow into the right lower lobe. Bronchoscopy by Mr. E. D. D. Davis showed the right lower bronchus to be occluded by œdema and granulation tissue. A probe was passed and a bead of pus escaped. Lipiodol was then injected through the bronchoscope. It was seen to well up and overflow. The skiagram showed that the lipiodol had again failed to enter the affected area. Bronchiectasis and foreign body being thus excluded, abscess was diagnosed, and it was decided to drain it. This was done by Mr. H. S. Clogg. The pleura was found to be non-adherent, and was treated in the way I have described. After operation all expectoration ceased and the boy made an excellent recovery. A sinus persisted, however, and it was decided to evulse the phrenic nerve. This also was done by Mr. Clogg. The cavity was obliterated, the sinus healed, and recovery was complete and permanent.

Anæsthesia.—This is all-important. I have never seen serious hæmorrhage, which used to be thought the great danger of lung incision, but I have seen disasters from aspiration of the infected material into the sound lung during operation. This is apt to occur if the pleural layers are not adherent, or if the cough reflex is abolished. Local anæsthesia is therefore the method of choice. If general anæsthesia is needed, then it is essential to have a highly expert anæsthetist who realizes the special problems of chest surgery, and intratracheal anæsthesia is, I believe, the right method.

If a chronic abscess cannot be cured by the methods described, or if, after cure of the abscess, bronchiectasis supervenes, then thoracoplasty may have to be considered.

Mr. A. Tudor Edwards: The efficient treatment of pulmonary abscess is dependent upon accurate diagnosis and exact localization of the lesion.

As abscess may arise from (1) precedent primary infection in the lung, (2) septic embolism secondary to infection elsewhere in the body, either spontaneously or post-operative, and (3) inhalations resulting from infection in, or operations upon, the upper respiratory passages, it will necessarily follow that any of these occurrences in the previous history will suggest the possibility of the lesion in the lung being infective in nature.

The physical signs are extremely variable, and, apart from localized dullness, may be absent. The symptoms consist of irritative cough with the expectoration of purulent sputum.

I shall not discuss either signs or symptoms in any detail: there are two general groups, one in which all the symptoms are intermittent and the other in which the symptoms are continuous. I draw particular attention to the second group, as the symptoms are indistinguishable from those of bronchiectasis, and unless complete investigation is carried out at a comparatively early stage, the period at which efficient treatment can be instituted is passed, and eventually a condition of secondary bronchiectasis is established. The abscess giving rise to the continuous symptoms generally results from inhalation.

X-ray examination.—This is essential in all cases of suspected abscess and invariably affords valuable information. The abscess, before rupture into a bronchus has occurred, appears as a somewhat irregularly rounded opacity in the clear lung field. In the inhalation types the opacity is more irregular and has a tendency to be rather triangular, with the apex towards the hilum and the base towards the periphery. After rupture into a bronchus, in the other type, a clear area is seen in the centre of the opacity which, when completely emptied, shows a fluid level altering with posture. It is essential to localize the relations of the abscess to the chest-wall by means of lateral and oblique radiograms.

Lipiodol introduced into the bronchial tree, fails to enter non-tuberculous abscesses, owing to the œdema and granulation tissue blocking the entrance thereto, and affords a means of differential diagnosis from bronchiectasis.

Medical treatment.—Medical treatment should always be given an opportunity before surgical treatment is advised. General hygienic and dietetic measures I do not propose to discuss, but there are two methods of treatment on medical lines which are of considerable value: (1) *Postural drainage*, which naturally can only be instituted after bronchial rupture has occurred. The optimum position for the evacuation of the abscess should be ascertained after repeated trials. The patient is then induced to occupy this position for gradually increasing periods two or three times a day.

Spirochætes of certain types are almost invariably found in the pus from pulmonary abscesses, and would appear to have a definitely deleterious effect in conjunction with other organisms; they may well account for the typical colour of

the pus. The beneficial effect of intravenous arsenic on spirochætal infections is well established, and although it is difficult to assess its value in these cases it should always be given. The elimination of all sources of sepsis elsewhere in the body should be carried out concurrently with the treatment of the abscess.

(2) *Artificial pneumothorax* would appear hypothetically to be the ideal treatment of an abscess which is incompletely draining into the bronchi. In those abscesses situated in the hilum it has a definite field of usefulness, but the diagnosis of a hilum abscess should be absolutely defined, by antero-posterior—and also by lateral—skiagrams.

During the last year I have operated upon three pulmonary abscesses which appeared radiologically, from the antero-posterior view, to be hilum abscesses, but the lung was widely adherent to the chest-wall posteriorly where the abscesses were situated and all had been treated for some time by artificial pneumothorax without benefit.

In the more superficial abscesses artificial pneumothorax is dangerous, owing to the risk of rupturing adhesions which overlie the abscess and attach that portion of lung to the chest-wall. This accident results in an acute septic pyothorax—a most serious complication. In five of my cases this complication was present.

Operative treatment.—(1) *Bronchoscopic drainage:* Drainage by the normal channels—the bronchi—is a most desirable treatment and surgically, appears to be best attained by bronchoscopic aspiration of the contents of the abscess. In practice, bronchoscopic lavage and aspiration have been disappointing, and, unless a gross foreign body is present, which is much more common in bronchiectasis than in abscess, would not appear to justify more than a short trial unless there is definite improvement in the patient's condition.

(2) *External drainage.*—External drainage should be undertaken in all cases of pulmonary abscess which fail to clear up by medical means, or in more definite language, any patient with a pulmonary abscess, who is not steadily improving both in general and local condition, should be submitted to operation. It is common to see general improvement in a patient while the abscess is passing from a subacute to a chronic condition and hence the alteration in the radiological appearances should be the deciding factor as to whether surgical measures should be instituted or medical treatment continued.

External drainage operations should always be carried out under local anæsthesia unless strongly contra-indicated by the mentality of the patient. In very nervous subjects, gas-oxygen, administered with positive pharyngeal pressure, in order to prevent aspiration, is the anæsthetic of choice. The upper lobe abscesses are often best approached through the upper axilla unless situated posteriorly; the lower lobe abscesses posteriorly, and those in the middle lobe through an anterior incision. The abscess cavity is emptied by posture one hour before operation. The operation should be done in one or two stages, according to the presence or absence of adhesions shutting off the main pleural cavity. A vertical incision is made over the site of the abscess down to the ribs and intercostal muscles. A portion of rib 2 or 2½ in. long is resected subperiosteally. When adhesions are present it is common to see some œdema and thickening of the posterior periosteum and parietal pleura. A hollow needle is inserted into the subjacent solid lung and aspiration performed. The removal of foul-smelling gas or pus is evidence of the needle-point being within the abscess cavity. The needle may be left *in situ* and the solid lung overlying the abscess incised with a cutting diathermy needle. In comparatively small abscesses a portion of the solid lung is removed, allowing adequate drainage with a soft-walled tube just projecting into the abscess cavity. In the larger abscesses a portion of rib above or below is removed, the intercostal vascular nervous bundle is ligatured and the intercostal muscles are removed. By this means most of the superficial wall of the abscess is exposed and can be removed by the cutting diathermy wire.

The whole wound is then lightly packed with paraffin gauze and healing is allowed to take place by granulation. Two-stage operation is undertaken when the pleura is free, and the first stage consists in removal of rib and intercostal structures overlying the abscess, but without incision of the parietal pleura. A pack of iodoform gauze is now laid against the pleura and the wound loosely sutured. Firm adhesions will form between the pleural layers in from seven to ten days, when the operation is completed similarly to the one-stage operation.

After-treatment.—Drainage is maintained until the discharge loses the purulent characters and is almost negligible in amount. It is essential to keep the superficial parts of the wound open until drainage is complete and the abscess cavity obliterated.

The inhalation abscesses appear to require much more prolonged drainage than the embolic types.

In cases in which diagnosis has been delayed, treatment instituted at a late stage, and secondary bronchiectasis already established, healing may take several months.

Phrenic evulsion.—This operation is of considerable value in the treatment of pulmonary abscess, for three reasons: (1) Occasionally, basal abscesses which have ruptured into the bronchus will heal completely with this operation without external drainage. (2) As I have previously pointed out, an early phrenic evulsion will frequently prevent the onset of secondary bronchiectasis and even cure early cases. (3) By relaxation of the lower parts of the lung, phrenic evulsion will hasten healing of large abscess cavities.

Thoracoplasty.—A clearer realization of the pathological changes in the lung secondary to the resolution of a pulmonary abscess should in many cases rid us of the necessity for extensive thoracoplastic procedures. The removal of a sufficient area of the chest wall at the time, or soon after drainage, will allow of localized collapse sufficient to neutralize the loss of pulmonary tissue in many cases. In others, early phrenic evulsion will be required to prevent traction upon, and subsequent dilatation of, the bronchi. Thoracoplasty will only be required in cases in which, as a result of late diagnosis or inadequate treatment, a generalized bronchiectasis supervenes, or in certain cases when secondary infection of the pleura has supervened.

In my opinion, lobectomy is never required for the single abscess. In the rarer cases of multiple abscesses localized to one lobe—not including multiple abscesses secondary to bronchiectasis—one type or other of this operation may be required.

My experience is limited to two cases in the group; in the first, healing by drainage took place after dilatation of the communications between the various pockets, and in the second the major portion of the middle lobe had been removed by diathermy in two stages. Lobectomy is required for bronchiectasis with or without extension of the ulceration into the lung parenchyma, and for diffuse suppurations. With the actual cautery I have been very disappointed; Souttar's steam cautery, which I have used on two occasions, is too slow in its action, but I am much impressed with the cutting diathermy, which I have been employing recently.

Forty-five cases of pulmonary abscess have been submitted to operation, comprising all types, from the gangrenous to the chronic simple abscess, but excluding those secondary to advanced bronchiectasis. In every case medical treatment has been given a definite trial; in all cases sufficient, and, in many cases, much too prolonged for the good of the patient. These cases therefore consist, as they should, of the failures of medical treatment.

In four of these forty-five cases the abscess was secondary to carcinoma of the lung. The patients still survive their drainage operations for periods varying from six weeks to four months. Nine patients have had a secondary infection of the

pleural cavity in addition to the pulmonary abscess, five—as mentioned before—owing to infection of a pneumothorax cavity. Of these nine, one died from toxæmia fourteen days after operation, three are recovering but still under treatment, and the remainder are well; one has a small residual sinus. Two patients with basal abscesses which were discharging incompletely into the bronchi, recovered completely, after phrenic evulsion. Of the remaining thirty, seven died (two from cerebral abscess, four from toxæmia, and one from aspiration pneumonia of the opposite side) and twenty-three recovered.

If the carcinoma patients, none of whom died as a result of operation, are excluded, eight patients out of forty-one have died—a mortality of 17·7 per cent.

Dr. L. S. T. Burrell: The treatment of lung abscess depends upon the position of the abscess and upon its character. As with an abscess in any other part of the body, treatment should aim at draining the pus. If the abscess is in communication with a bronchus and the pus is being freely expectorated, spontaneous recovery frequently takes place; on the other hand, chronic abscess usually results, so that surgical treatment becomes necessary at a later and more difficult stage. In the majority of cases the lung abscess follows operation on the mouth, nose or throat, especially tonsillectomy, but the possibility of foreign body should always be borne in mind.

If the abscess communicates with a bronchus, the safest procedure is to collapse the lung by means of artificial pneumothorax, as soon as a diagnosis is made. If it is found that adhesions have already formed or that the abscess is superficial, there is considerable danger of a rupture into the pleural cavity producing a pyo-pneumothorax. For this reason artificial pneumothorax should be discontinued if the pleura is adherent over the abscess, and drainage by open operation should be employed. The hypothetical objections to artificial pneumothorax are:—

(1) That by blocking some of the bronchi, drainage is prevented or, at any rate, hindered. (2) That although the lung is collapsed, the wall of the abscess is not, so that no effect on the abscess is obtained. (3) The danger of rupture into the pleural cavity is considerably increased. In my opinion these objections apply only to those cases which are left too long on simple expectant treatment before a pneumothorax is induced. If a case is first seen many months after the abscess has formed, pneumothorax is hardly ever of any value and in most cases is actually dangerous. On the other hand, if carried out in the very early stages, it will prevent an abscess from spreading through the lung tissue and one will hardly ever see a small abscess develop into a large infiltrated area of pus if the lung is collapsed sufficiently early; in other words, it limits the spread of the abscess and, in my experience, the majority of these cases do extremely well with early collapse. If the abscess is superficial or if there is no communication with the bronchus, rib resection and drainage should be employed. In these cases it is essential first to localize the exact position of the abscess, and secondly to see that firm adhesions exist between the visceral and parietal pleura over the region to be operated upon. For this reason it is usually better to perform the operation in two stages, the first merely to fix the visceral pleura to the chest wall and encourage adhesions around. In order to localize the position of the abscess, the safest procedure is to have skiagrams taken in various directions. I have found lipiodol of little value in these cases as it does not enter the abscess cavity even if there is free communication with a bronchus.

In the case of a chronic abscess more serious surgical methods may have to be adopted, and probably the operation of the future will be lobectomy.

I should like, in conclusion, to emphasize the fact that the success of pneumothorax depends upon its early application and that, therefore, I do not agree with the initial so-called "expectant" treatment. If the initial stage is passed before the patient is seen, then there are grave dangers associated with pneumothorax, and expectant treatment may be tried before resorting to surgical operation.

Mr. J. E. H. Roberts: We have so far been discussing lung abscess without clearly differentiating between the various types. All the radiograms which have been shown relate to a single cavity, with well-defined outline, mostly showing a fluid level. In practice the clinical diagnosis of lung abscess is made in the following conditions: (1) Frank gangrene of part of a lung without a cavity. (2) A single abscess cavity, the wall of which is composed of gangrenous lung tissue. (3) Multiple small abscess cavities in spongy lung. (4) A single more chronic abscess cavity lined by granulation tissue. (5) A single abscess cavity lined by epithelium, generally of the stratified squamous variety. This may result, as in five of my own cases, from implantation of skin driven into the lung by a missile, or, in non-traumatic cases, by metaplasia of ciliated epithelium from a bronchus. (6) So-called inter-lobar empyema, which is nearly always an abscess lined by pulmonary tissue, not by pleura, and communicating with several bronchi. (7) Localized bronchiectasis, which, even with the aid of lipiodol, radiograms, may be impossible to distinguish from abscess. (8) Bronchiectatic abscess. (9) Abscess due to bronchial obstruction by a foreign body or neoplasm. No uniform method of treatment can be laid down for these various conditions, and the prognosis is so different that no statistics of the result of treatment are of value unless the actual condition present is clearly indicated.

I should like to emphasize the danger of exploratory puncture of the lung when a superficially placed abscess cavity is present. I know of two cases in which this procedure was followed by a stinking pyopneumothorax and death.

With regard to treatment, I have only time to take up one or two points.

Firstly, as a certain proportion of patients recover under medical treatment, I believe that this should always be carried on for a time. I show skiagrams from the case of a man with a large circumscribed abscess, with a fluid level, who had been bringing up stinking sputum in increasing quantities for four and a half months. I was asked to drain the abscess, but there was six weeks' delay in admitting him to hospital, during which time he was treated as an out-patient in the creosote chamber and by inversion. On admission he was found to be free from sputum, and a new radiogram showed a normal lung. Another somewhat similar case occurred in a boy, aged 15.

While I believe there is a time for medical treatment, I feel that in a large percentage of cases this is carried on far too long and operation is delayed until much too late a stage, though I am not prepared at present to lay down rules as to how long medical treatment should be persisted in.

I was at first greatly attracted by the possibilities of artificial pneumothorax, but having had to drain a resulting pyopneumothorax in four cases during the last six months, I now believe that, in the case of a superficial abscess, it is too dangerous and should not be performed. If performed at all, it should be restricted to deep-seated abscesses near the hilum. I agree with previous speakers that drainage operations should be carried out in two stages, and that the lung should be opened with the diathermic point or actual cautery, preferably the former. I prefer actually to remove a circular piece of lung with the cautery, in order to provide a drainage which does not necessitate the introduction of drainage material into the lung.

One speaker has mentioned resection of a rib. I prefer to resect portions of at least two, and often three, ribs. If only one rib is resected it may appear that there are good adhesions enabling a one-stage operation to be done, whereas in reality these adhesions exist only in a narrow band and a one-stage operation is likely to be followed by infection of the pleura.

Mr. Herbert Tilley said that aspiration of pulmonary abscesses, when these could be reached by direct bronchoscopy, was a valuable aid in treatment.

[Mr. Tilley showed some patterns of tubes now in use in Chevalier Jackson's clinic in the Jefferson Hospital, Philadelphia. These tubes had straight or curved

flexible spiral wire terminals which enabled any of the pulmonary lobe bronchi to be explored under the guidance of direct vision.]

In Dr. Jackson's clinic, no anæsthetics, local or general, were used in the case of children, but occasionally for adults, a 10% solution of cocaine was applied to each pyriform sinus about fifteen minutes before the bronchoscope was passed. The resulting anæsthesia allowed the instrument to be passed through the glottis with the minimum of discomfort.

[Mr. Tilley described some of his recent experiences in Dr. Jackson's bronchoscopic clinic and his visits to the wards where it was possible to see the results of the treatment of pulmonary abscess and other pathological conditions of the lungs, e.g., bronchiectasis, bronchial strictures, and even chronic bronchitis and asthma.]

In cases of asthma, great relief was afforded by a bi-weekly aspiration of tenacious mucoid secretions from the lobar bronchi. Much stress was laid by Dr. Chevalier Jackson on aeration and drainage of the lungs by which means the cilia were enabled to resume their function for increasingly long periods. All the patients were anxious to have even the temporary comfort which aspiration by the direct method afforded, and in many cases the apparently permanent improvement was very striking.

Pulmonary complications following tonsil operations seemed to be much more common in the United States than in this country, probably because many of the surgeons operated with their patients in the sitting position or with the head and shoulders raised. In these circumstances, blood, septic material, or portions of tissue were almost certain to gravitate towards the larynx and trachea in spite of every precaution to prevent such a descent. In this country, it was an almost universal custom to have the patient lying on his back with the head over-extended, so that the blood drained backwards into the naso-pharynx and out from the nostrils. He knew that others held the view that the pulmonary complications following tonsillectomy were caused by the detachment of septic thrombi into the circulation, from the venous radicles in the tonsillar recess. If that were so, the question arose, "Why are such post-operative pulmonary lesions so comparatively rare in this country, when all post-operative tonsillar recesses are equally septic?" If surgeons would have sufficient patience to become skilled in the use of the bronchoscope, they would find that it was no longer a toy, but an extremely useful addition to their armamentarium in dealing with many pathological conditions of the lungs, even when these were not the result of entry of a foreign body into the lower air passages.

Dr. H. V. Morlock said that his senior colleague, Dr. A. J. Scott Pinchin, and himself had recently reviewed twenty-nine cases of lung abscess seen at the City of London Hospital for Diseases of the Heart and Lungs, Victoria Park. Of these, fifteen had been treated on medical lines (postural drainage, etc.). Thirteen patients had recovered. Two had died: one from repeated hæmorrhages, one shortly after admission to hospital.

With regard to the question as to when medical treatment should give place to surgical measures, they had arrived at the opinion that a period of from two to three months' medical treatment should be allowed. If during this period the patient was showing no signs of improvement or was going downhill, surgical measures should be adopted earlier. Five cases had been treated by artificial pneumothorax, two patients recovered; in two cases pyopneumothorax had developed, but a complete recovery had followed drainage of the empyema. One patient had died from pyopneumothorax. Two cases were treated by thoracoplasty; one patient had died, the other had recovered after drainage of an empyema which developed after the operation. Six cases were treated by incision and drainage; three patients had recovered, one had improved but died later from pneumonia in the other lung, two had died after operation.

Mr. R. H. O. B. Robinson said that in order to emphasize a point made by Mr. J. E. H. Roberts, he would record a case on which he had operated at Victoria Park Hospital. An abscess as large as a fœtal head had been present in the lung, but the two layers of the pleura had only adhered over a very narrow strip, so that a one-stage operation was quite unjustifiable. He would also point out the danger of spreading infection in the thoracic parietes. Fatal results followed drainage of abscesses, in which gangrene of the surrounding lung was present, and he suggested that in these cases, two-stage operations should always be carried out to shut off the connective tissue and muscle planes by granulation, before opening into lung tissue.

Dr. W. E. Lloyd said he had been impressed by the fact that in nearly all the skiagrams shown, the abscess was present in the right lung. This was in keeping with the accepted view that in many cases the abscess was due to inhalation of septic material, secondary to tonsillectomy or other operation on the upper air passages.

With regard to the diagnosis of pulmonary abscess in elderly patients, he thought that neoplasm of the lung should be considered. Evarts Graham, in a recent paper, had stated that out of 330 cases of pulmonary suppuration, excluding empyema and tuberculosis, thirty-three (10%) of the cases were due to carcinoma of the lung.

One other point in diagnosis was worth mentioning. In the early stages of lung abscess, foul expectoration was often absent, but a very suggestive symptom in such cases was occasional fœtor of the breath.

Mr. A. Dickson Wright: The condition of pyopneumothorax has been mentioned as a complication of lung abscess. The gas in the pleural cavity may be derived from one of three sources: (1) Introduced in production of artificial pneumothorax. (2) From a bronchus communicating with the abscess cavity. (3) From the gas-forming spore-bearers and spirochætes in the pus of the abscess. As illustrating the last type, I recently saw a case of empyema in which, in addition to signs of fluid, there were also those of pneumothorax. The patient was a woman, aged 52, extremely ill from toxæmia and dyspnœa. Aspiration yielded a pint of the most offensive pus that I had ever encountered, so offensive that the aspirating cannula, after being boiled for three hours, still smelled abominably, and all the other articles in the sterilizer were polluted. A bacteriological examination revealed spirochætes and anaerobic bacilli. Aspiration was persisted in every second day, although the physician in charge of the case was anxious that thoracotomy and drainage should be done. On each subsequent aspiration smaller quantities of pus were obtained, and the fœtor of the pus diminished. On the sixth aspiration only clear fluid was obtained, and a skiagram no longer showed a fluid shadow. The method of aspiration was the excellent one originated by Mr. Tudor Edwards: an incision down to the parietal pleura was kept open by a gauze plug, and repeated aspirations were performed painlessly through this wound. Anti-gas gangrene serum and met-arsenobenzol were also given. After the sixth aspiration the temperature remained level for a week, and then began to rise again. It was inferred that this was because the superficial lung abscess—which, in rupturing into the pleural cavity, had caused the empyema—was now again a closed space as a result of the formation of pleural adhesions, and absorption of toxins was again occurring. It was decided to wait for possible rupture into a bronchus before operating upon the abscess. This occurred about seven days later and after a few days' expectoration of foul pus, the pyrexia diminished and the patient convalesced; she left hospital a few days ago. This case illustrates the value of expectant treatment in dealing either with the abscess or with the stinking empyema produced by it. No matter how toxic the patient's condition, treatment on these lines, though it should not be persisted in too long, may result in a complete cure, or at least will get the patient into better state for operation.

Clinical Section.

[*February* 14, 1930, *continued.*]

? Keloid of the Thoracic Wall.—HAROLD EDWARDS, M.S.

Male, aged 44 years, bus driver. On the chest wall below the left clavicle is a scarred area 2½ in. by 2 in. The scar has infiltrated the whole thickness of the skin but is unattached to the deep structures. There are some outlying erythematous nodules, one of which is of considerable size, forming a wart-like mass. This has ulcerated.

The patient first noticed the condition about seven years ago. Ulceration of the wart-like mass dates back six months. The patient attributes the ulceration to friction by braces. Wassermann reaction negative.

Treatment so far has been the application of 50 mgm. of radium for two hours. This took place five weeks ago, and has produced no apparent change.

Discussion.—Dr. W. J. CARR (President) said he wondered if there was not a considerable risk that the wart might become malignant; if so, should it not be freely removed forthwith?

Dr. PARKES WEBER said that after excision of a keloid, a linear keloid developed in the excision wound, with a row of small punctiform keloids on each side, marking the sites of the suture punctures. He had seen this follow excision of vaccination keloids.[1] Indeed, that was the best test for the genuineness of a keloid. The best treatment was probably the application of X-rays, but keloids tended to atrophy spontaneously in time.

Schlatter's Disease.—R. RUTHERFORD, L.R.C.P., M.R.C.S.

W. D., male, aged 14. Knees were painful after patient played football eight months ago. Tubercles of tibiæ are enlarged, right more than left. Tubercle of right tibia feels cystic. A skiagram clearly shows the fragmentation of the tibial tubercle.

Familial Multiple Exostoses: Father and Daughter Affected.—R. RUTHERFORD, L.R.C.P., M.R.C.S.

E. M., male, aged 43. Has both pedunculated and sessile exostoses growing from the lower ends of both femora, and sessile bony growths on the heads of both fibulæ. The lower left costal cartilages show some irregularities near their junctions with the ribs. On the mother's side of the family there is a history of these bony outgrowths appearing in certain members every generation.

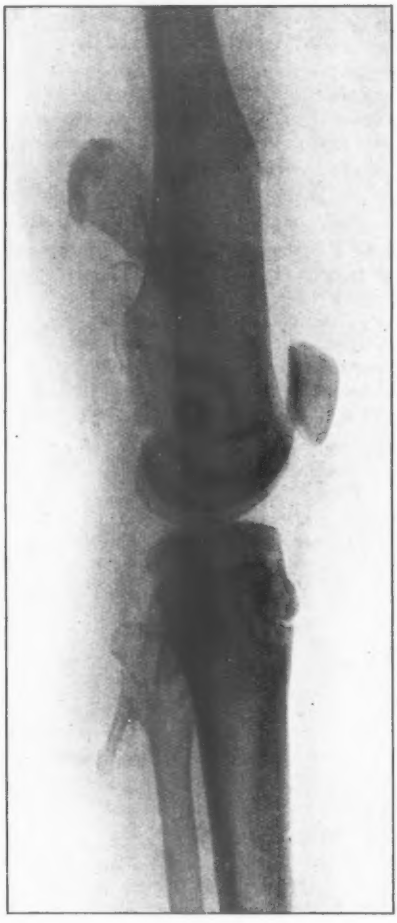
M. M., aged 14, daughter of E. M., also shows multiple exostoses, as follows:—

Left half:—(1) Third rib near its cartilage. (2) Vertebral border of scapula. (3) Lower end of radius. (4) Lower end of femur outer and inner sides. (5) Upper and lower ends of fibula. (6) Proximal phalanx of middle finger, near base. (7) Proximal phalanx of thumb, near base.

Right side:—(1) Lower end of femur, inner side. (2) Proximal phalanx of ring finger, near base.

It is noteworthy that there is a left-sided preponderance in the distribution of these exostoses and that the daughter has a sacralization of the last lumbar vertebra on the right side.

1 I have also seen keloids resulting from Pirquet's cuti-reaction for tuberculosis as well as after vaccination against smallpox (F. P. W.)



Familial Multiple Exostoses.

M, M., aged 14. (Mr. R. Rutherford's Case.)

Where the exostoses occur on the same bones in these two cases it is remarkable with what fidelity they conform in position and appearance.

The girl was breast-fed and had a normal infancy in every way. The skiagram shows the pedunculated and sessile growths on her femur and fibula.

Case of the Jaw-Winking Phenomenon.—HAROLD AVERY, M.B.

I. K., male, aged 30, a Russian Jew, has had since birth a partial ptosis of the left upper lid, which, however, rises completely when the mouth is opened. As the jaws work up and down in chewing, or from side to side, the left upper eyelid moves up and down so that he appears to be winking. Physical examination reveals no

other abnormality. One paternal uncle is similarly affected on the left side, but no other member of the family is so affected.

Since Marcus Gunn showed the first recorded case of the Jaw-Winking Phenomenon before the Ophthalmological Society of the United Kingdom, in 1883, about a hundred cases have been reported. The majority have been in males, and the condition has generally been unilateral, the left being affected in 80% of the cases, both sides in four cases. As a rule, the phenomenon is congenital—rarely familial.

Dr. E. A. COCKAYNE said he had seen a case of the kind in a small baby. When the child sucked, the eyelid was lifted and the white of the eye could be seen. He had ascertained some years later that the condition still remained, and that it happened when chewing was carried out. It had been more noticeable in that case than in the present one. He (the speaker) had collected the cases published in the literature and published them in the *British Journal of Children's Diseases*, 1914, xi, 352.

Case of Rumination in Man.—HAROLD AVERY, M.B.

H. P., male, aged 53, a Russian Jew, has noticed since childhood that food returns into his mouth after meals, without nausea or discomfort. It tastes the same as when first taken, and after rechewing and enjoying it he swallows it again. Rumination generally continues for two or three hours after a meal, the food coming back in from fifteen to twenty mouthfuls. As soon as the food begins to taste sour or bitter, rumination ceases. If he falls off to sleep after a meal he finds he is unable to bring up the food after from half to three quarters of an hour. He takes great pleasure in his power of enjoying his meals over again, but is embarrassed in company and tries to hide the action. He knows of no other person who has the same power. Physical examination reveals no abnormality.

X-ray examination showed the barium bolus passing extremely rapidly down a slightly dilated œsophagus into a normally shaped stomach. When rumination occurs the patient swallows, the upper segment of the stomach contracts, and part of the contents pass up through the œsophagus into the mouth. This only occurs if the stomach is full; with smaller quantities a part of the gastric contents rises into the œsophagus but does not reach the mouth. No peristalsis or reverse peristalsis was seen.

Discussion.—Dr. COCKAYNE said he remembered going to the Finsbury Park Empire with Dr. Parkes Weber and seeing a Frenchman who could swallow frogs and goldfish, and would, on request, produce again from his stomach either a frog or a goldfish still alive. He had previously drunk a large quantity of water. Dr. Parkes Weber had taken litmus paper with him, and on testing the water returned had found that it was not acid. The performer's father had been able to do the same thing.

Dr. PARKES WEBER said that in cases of human rumination it would be interesting to test the gastric contents after a test breakfast by the modern fractional method, to see if the quantity of free hydrochloric acid was below the normal or up to the normal. He (Dr. Weber) thought that the explanation of cases supposed to be examples of rumination secondary to organic gastric disease, was to be sought in the occasional occurrence of organic disease of the stomach in individuals who previously had either ruminated or possessed the latent power of rumination.

Dr. E. STOLKIND said that Desternes had recorded a case of a man who had ruminated since childhood. When at school he simulated sickness by ruminating and so obtained a holiday. He exhibited the phenomenon to the other boys to amuse them. On one occasion he swallowed a small fish and returned it later.

With regard to the point that these patients might also have organic disease of the stomach: he had himself treated two cases of gastric ulcers in which there had been rumination. One, a man aged 43, had in addition nervous rhinitis; the other only had rumination when excited.

As rumination might be developed by mere imitation it became important, for instance, in the case of a school teacher, to insist on the necessity for treatment (mostly psychotherapeutic).

Advanced Osteo-arthropathy in a Case of Pulmonary Neoplasm.—HAROLD AVERY, M.B.

C. B., male, aged 38, reported at Charing Cross Hospital in August, 1929, with a three weeks' history of pain and swelling in the knees and ankles, and enlargement of the finger-tips. The arms and elbow-joints were also beginning to ache. He was losing weight (8½ lb. in four months), felt weak, and tired easily. No cough or dyspnœa; expectoration slight, mornings only and never tinged with blood. Appetite good; no night sweats. There was a past history of phthisis for which he had been treated at Brompton Hospital in 1907, but had discharged himself and had received no treatment since. Past history.—Scarlet fever in 1910. Denies syphilis. Father died from pulmonary tuberculosis; a brother died whilst on war service, from influenza; the rest of the family are healthy.

Examination revealed advanced clubbing of the fingers. The wrist- and ankle-joints were swollen, slightly tender, painful on movement, not red or hot. The knees were slightly swollen, the other joints normal. On examination of the chest there was a small area of decreased breath sounds at the inner end of the first and second intercostal spaces anteriorly. No other abnormality was found on physical examination.

Sputum was T.B. negative on several occasions. Wassermann reaction, negative. Skiagram showed a dense area of opacity in the left upper lobe: ? old fibrosed tuberculous patch, ? neoplasm. The fingers and wrists showed the changes of pulmonary osteo-arthropathy. Treatment consisted of rest in bed, cod-liver oil and tuberculin B.E.

The condition was unchanged on discharge in September, 1929. In October, 1929, impairment of note over the upper part of the left lung posteriorly became apparent, with decrease of the breath sounds. The skiagram showed enlargement of the shadow which now had the definite appearance of a neoplasm. Deep X-ray therapy was began by Dr. Russell-Reynolds.

In December the neoplasm seemed to be larger. During January and February, 1930, the patient has been having deep X-ray therapy from Dr. Finzi, and the growth appears to be slightly smaller.

Osteitis Deformans with Sarcoma.—A. E. MORTIMER WOOLF, F.R.C.S.

C. G., male, aged 55 years, married.

History.—Complains of an egg-like lump on the back of the upper part of the left thigh, present since August, 1929. It began as a small nodule and has gradually increased in size, but has caused no interference with his work.

He had no illnesses or complaints until five years ago, when he had an attack of bronchitis, for which he was not confined to bed.

Four years ago he noticed that his hats were tight and he has been taking increasingly larger sizes in headgear ever since.

Shortly after this he had an occasional "shooting" pain in his left leg, from above downwards. Two years ago he noticed for the first time that he was bow-legged, but this deformity has not interfered with his work.

Has had occasional headaches, relieved by aspirin.

Has lost 4 lb. weight during the last twelve months. Has not noticed any loss of stature. Kept at work until October, 1929, when he first sought advice at St. Bartholomew's Hospital.

Family History.—Father died from heart disease; mother killed in an accident; one brother alive and well; five children alive and well.

Urine.—Trace of albumin; no Bence-Jones protein.

Skiagram.—Sella turcica normal. The bones, including those of the skull, show evidence of osteitis deformans.

Section of Anæsthetics.

[*February* 7, 1930.]

DISCUSSION ON ANÆSTHESIA IN THORACIC SURGERY.

Dr. C. Langton Hewer: I suppose that few branches of therapeutics have advanced further during the last fifteen years than thoracic surgery has done; no doubt the European war is largely responsible for this result. Previous to 1914 an intrathoracic operation was regarded as distinctly risky, whereas at the present time an exploratory thoracotomy can be performed with almost the same ease and safety as an exploratory laparotomy. This diminution in risk has been brought about, in part at least, by improved anæsthetic technique.

Before discussing the actual anæsthetic problems involved, a few words might be said about the preliminary preparation of the patient. There is now general agreement as to the undesirability of drastic purgation before operation, but I think we may go further and ensure that our patients have an abundance of fluid in their systems before coming to the operating theatre. I am convinced that the simple process of directing patients to drink as much fluid as they conveniently can between meals for two or three days before operation, makes a great difference to their condition afterwards. It is noticeable how comfortable and free from vomiting and acidosis are the patients who have been prepared in this way; the dry tongue and general parched feeling, with thick and scanty urine, so often found after prolonged operations, are markedly absent. It seems reasonable to suppose that if the fluid intake before operation is large, the toxins which are liberated during the traumatization of tissues will be diluted and thus have less effect upon the nervous and circulatory systems. It is possible that glucose given in the fluid will effect even better results, but personally I am not convinced of this. For most adult patients a preliminary hypnotic is desirable, but large doses should be avoided or the coughing reflex may be unduly weakened. A fairly healthy man should not have more than morphine gr. $\frac{1}{4}$ and hyoscine gr. $\frac{1}{100}$, whilst for a woman gr. $\frac{1}{6}$ of morphine is usually sufficient. If no ether is to be used, atropine is unnecessary and merely makes the patient feel uncomfortably dry. Finally, in chest cases, it is a mistake to begin the actual induction of anæsthesia before the surgeon is ready to begin operating. It is quite possible for from ten to fifteen minutes to be wasted at this stage, to the detriment of the patient's condition.

At this point the after-treatment of thoracic cases may be conveniently considered. It has been shown by Dr. J. Alexander, of Ann Arbor, Michigan, that a larger quantity of saline at a higher temperature can be given rectally to an anæsthetized patient than if it is left until consciousness is regained. At the Brompton Hospital and at Queen Mary's Hospital, Roehampton, in Mr. J. E. H. Roberts' chest cases 1 litre (about $1\frac{3}{4}$ pints) of saline, containing 5% glucose, is given while the chest wall is being closed. The temperature of the fluid in the funnel is 115° F., and this injection is practically always retained. The patient thus reaps the advantages of fluid, heat and glucose, and this treatment partly explains the satisfactory condition after quite severe operations. While the patient is on the table, his bed and room should be made thoroughly hot. It seems impossible to

over-estimate the importance of heat in treating post-operative shock. It is fairly easy to provide a warm atmosphere in a small room, but almost impossible in a large ward. For this reason I think that all hospitals should be provided with recovery rooms where patients can remain after operation until their temperature is normal and their condition satisfactory. If the patient must be taken to a general ward, an electric cradle, or one of the new electrically heated blankets, is much more effective and less dangerous than hot-water bottles.

We may now pass on to a consideration of the anæsthetic problems which thoracic operations present. In the first place, patients are usually in poor general condition. They may be suffering from a great variety of complaints, such as phthisis, bronchiectasis, empyema, pulmonary abscess, or tumour, all of which tend to depress health, apart altogether from the local lesion. Secondly, the chest wall is vascular, and extensive operations inevitably entail considerable oozing from large surfaces, with corresponding shock. Again, proceedings which involve the opening of one or both pleural cavities introduce a series of factors dependent upon the disturbance of the normal pressure relationships of the thoracic viscera. Lastly, serious respiratory obstruction may arise from the pressure of tumours upon the air passages, or from the presence of blood or pus within their lumen.

It follows, then, that for thoracic surgery, a type of anæsthesia is required which should have the following characteristics:—

(1) It must have no depressant effect upon the body as a whole, even after prolonged administration. (2) It must have no harmful effect upon normal or diseased lung tissue, and must not increase the secretion of the respiratory passages. (3) It must not abolish the coughing reflex, so that fluids in the air passages can be evacuated both during and immediately after operation. (4) It must be followed by rapid recovery without undue after-effects. (5) It must enable one or both pleural cavities to be opened without danger should this be necessary. (6) It must ensure adequate oxygenation of the blood, even when normal respiration is obstructed by tumour or other cause.

Let us consider how the available methods of anæsthesia conform to these requirements.

Local Analgesia.—This has a useful place in thoracic surgery, a combination of infiltration and paravertebral dorsal block being commonly employed. In the present state of our knowledge I do not think that high spinal block is justifiable for chest operations. The advantages of local analgesia are that it has no effect upon lung tissue or upon secretions, that the operative field is comparatively dry and that after-effects are rare. After-pain, on the other hand, is definitely greater than with general anæsthesia, especially in cases in which the intercostal nerves have been blocked. The coughing reflex is not abolished, but the effectiveness of coughing is diminished for a considerable time after intercostal block, owing to the temporary paralysis of the abdominal muscles. Comparatively minor operations—such as phrenicotomy, the drainage of pleural and pericardial effusions, the induction of artificial pneumothorax and resection of rib for acute empyema—can often be conveniently performed in adults under infiltration analgesia only. It must not be forgotten, however, that there is such a condition as "pleural shock," which may give rise to alarming collapse. In extensive operations, local analgesia has definite disadvantages. The injection of large quantities of novocaine and adrenalin solutions may give rise to severe toxic symptoms. It is possible that the adoption of the new synthetic drug "percaine," may diminish these in the future, owing to the extreme dilution in which it can be used. There have recently been several cases of sudden death during the injection of novocaine and adrenalin. I believe that these have been caused not by an overdose of the drugs, but by an inadvertent intravenous injection. It has been shown repeatedly that a minute amount of novocaine and adrenalin injected into a vein may cause heart failure,

and I believe that the practice of simply injecting solution without ever performing the aspiration test, and without seeing that the needle point is always moving, has led to these fatalities. There are several types of continuous suction syringe on the market which are convenient for infiltrating large areas, as the barrel of the syringe need not be detached from the needle for refilling, but they do not allow the aspiration test to be carried out, and are therefore dangerous when infiltrating vascular areas, such as the thoracic wall. It is noteworthy that Finsterer and Farr, who have both had wide experience with local analgesia, have recently stressed the dangers of intravenous injection. A disadvantage of local analgesia in thoracic surgery is that no protection is afforded against respiratory obstruction, or against collapse of the lung when the pleural cavity is opened. This method is inadvisable in the case of young children, and when injections would have to be made through septic areas. If local analgesia is decided upon, it must be remembered that the actual injection of the fluid is only the beginning of the anæsthetist's duties. In extensive operations the patient requires just as careful attention as when a general anæsthetic is being used. For example, it is essential to recognize and treat the initial stages of shock, cyanosis may require oxygen inhalation, whilst the unexpected opening of the pleural cavities may necessitate the immediate application of differential pressure.

Chloroform.—This is not irritating and does not cause increased secretion of saliva and mucus, nor is the depth of respiration appreciably increased. It has a definite place in thoracic surgery, being especially useful in inducing anæsthesia in patients suffering from a high degree of respiratory obstruction when it is necessary to avoid any congestion and any increase in the depth of respiration. Chloroform, however, has several objectionable features. It shares with ether the defect of producing anæsthesia by combining chemically with the lipoid constituents of nerve-cells, so that it is eliminated slowly and may give rise to after-effects such as prolonged vomiting. Again, it is generally accepted that light chloroform anæsthesia is not without the danger of primary cardiac failure, while if full surgical anæsthesia is maintained, the coughing reflex will be abolished during operation, and for some time afterwards.

Ether.—This allows a very light anæsthesia to be maintained without the risk of primary cardiac failure. It is often useful when given in this way by the open method to children who are too small to be anæsthetized satisfactorily with nitrous oxide and oxygen. The majority of these will be cases of acute empyemata which only need short operations, and they almost invariably do well. For more extensive thoracic operations, however, ether is not satisfactory. It is liable to act as an irritant to the respiratory passages, and may lead to an increase in their secretions. Again, post-anæsthetic vomiting is common with ether, and a pronounced fall of blood-pressure is liable to occur after a long administration. These disadvantages can be overcome to a certain extent by using a minimal amount of ether combined with nitrous oxide and oxygen in endotracheal insufflation, by an adequate preliminary injection of atropine and by subsequent " de-etherization " with carbon dioxide and oxygen. Administered in this way, ether is of service in cases of respiratory obstruction.

Paraldehyde and Avertin.—The more recent rectal techniques using these drugs necessitate a post-operative period of depressed or absent reflexes, and this is definitely undesirable in thoracic cases. In my opinion the retention of an unimpaired coughing reflex is of the highest importance.

Nitrous oxide-oxygen.—This anæsthetic is in an entirely different category from any of the foregoing. It is probably true that nitrous oxide has no chemical effect, but acts physically, by limiting the oxygen supply to the cells of the cerebral cortex. At any rate, it appears to have no deleterious effects whatever upon either normal or diseased tissues. It is interesting to know that animals suffering from

pulmonary tuberculosis have been anæsthetized with nitrous oxide and oxygen for forty-eight consecutive hours without any exacerbation of the disease being observed. The only way in which this anæsthetic can produce untoward effects is by partial asphyxia, if the oxygen percentage is unduly cut down. The advantages of nitrous oxide-oxygen for thoracic surgery are many: It is non-irritating to the respiratory passages, and causes no increase in the secretions of saliva and mucus; the coughing reflex is not abolished; the recovery time is extremely short, whilst after-effects are conspicuous by their absence. If vomiting occurs more than once immediately after the patient regains consciousness, it is usually due to some other cause than the anæsthetic, for example, the preliminary morphine-scopolamine injection. Finally, if the gases are administered under pressure, one or both pleural cavities can be opened without the occurrence of dyspnœa. The only real disadvantage of nitrous oxide-oxygen is the poor muscular relaxation. This, however, is of very little importance in thoracic surgery. It will be seen, therefore, that of the anæsthetics so far considered, nitrous oxide-oxygen is the one most generally suitable for major thoracic work, and I have no doubt that its recent adoption has done more than any other one factor to render these operations the safe proceedings which they are to-day.

Ethylene and oxygen.—Ethylene can be used in the same way and with the same apparatus as nitrous oxide. It has two advantages over the former gas. Firstly, a slightly higher percentage of oxygen can be employed, and secondly, the muscular relaxation obtained is slightly greater. I have only used ethylene in about 130 cases, but in my opinion these slight gains are more than offset by three serious drawbacks. The commercial gas has a most unpleasant smell, and its administration is more likely to be followed by nausea and vomiting than is nitrous oxide. Again, the mixture used is highly explosive, and is definitely dangerous if employed in the X-ray room or when lung tissue is being cauterized.

Acetylene and oxygen.—Acetylene is largely used in Germany under the name "narcylene." I have had no experience with this gas in anæsthesia, but apparently it is very similar to ethylene.

Having mentioned the different anæsthetics at our disposal, let us consider the commoner thoracic operations and the best method of anæsthesia applicable to each case.

For anæsthetic purposes we may divide thoracic operations into the following seven groups:—(1) Superficial operations upon the thoracic wall. (2) Extrapleural operations involving the whole thickness of the thoracic wall. (3) Operations involving the opening of one pleural cavity. (4) Extensive thoracotomies which may necessitate the opening of both pleural cavities simultaneously. (5) Intrapulmonary operations. (6) Cases in which a bronchial fistula is present. (7) Cases in which some obstruction to the respiratory passages is present.

(1) *Superficial operations upon the thoracic wall.*—The commonest procedures in this group are those of excision and needling of carcinomata of the breast. These call for no special comment, as the cases almost invariably do very well with pure nitrous oxide and oxygen anæsthesia.

(2) *Extrapleural operations involving the whole thickness of the thoracic wall.*—The most usual operation in this group is thoracoplasty, which is generally employed for phthisis, bronchiectasis or chronic empyema. The chief difficulties with which the anæsthetist has to contend are the lateral position with the patient lying on his sound side, the shock which attends the stripping of the periosteum from the ribs, the hæmorrhage, which is rarely negligible, and, in bronchiectasis cases, the continual expectoration of muco-pus. I have no doubt that the best anæsthetic is pure nitrous oxide-oxygen. The difficulty of the position is largely obviated by using the useful rubber harness devised by Mr. R. J. Clausen which enables the face-piece to be kept firmly in position, however awkwardly the head

may be placed. In bronchiectasis cases it is a great help to make the patient lean over and cough up all loose sputum before beginning with the anæsthetic. Shock appears to be most easily estimated by taking the pulse-rate together with the pulse pressure. If it seems to be becoming excessive, the surgeon should be informed without delay, and the rest of the operation should be deferred to a later stage. That this can usually be done if the patient's condition is poor, constitutes one of the advantages of thoracic over abdominal surgery from the anæsthetist's point of view. Thoracoplasty cases anæsthetized in this way do remarkably well considering the severity of the operation. Consciousness returns within a few minutes of the cessation of the anæsthetic, and after-effects are rare. I am assured by a ward sister that it is not uncommon for a patient to have a thoracoplasty done at 11 a.m. and to have an ordinary lunch at 1 p.m. Chiefly through the kindness of Mr. W. E. Underwood, one of the medical officers at Queen Mary's Hospital, Roehampton, I have been able to obtain records of the last 100 thoracoplasties which I have anæsthetized with nitrous oxide-oxygen. In this series there was only one operative death, which occurred in a patient suffering from chronic empyema who was in a poor condition. He recovered normally from the anæsthetic and gave no anxiety until about ten hours later when he collapsed. He died from heart failure about twenty hours after operation. Looking back upon this case, I blame myself for letting the surgeon do too much in one stage. I do not believe that the anæsthetic was in any way responsible for the death. The remaining ninety-nine patients all did well from the anæsthetic point of view. Another type of operation coming within this group is that of cardiolysis for pericardial adhesions. This is really only a partial thoracoplasty and can be treated as such, always remembering that the oxygen percentage must not be unduly cut down, as the heart is already handicapped. A different type of operation which was almost unknown, but which is now becoming increasingly common, is the treatment of œsophageal carcinomata with radium or by excision. The surgical approach is usually from the back, and necessitates the patient lying on his face. If the anæsthetist uses the double endotracheal method it is rare for difficulties to arise. It is always wise to plug gauze tightly round the tubes immediately above the glottis, in case severe hæmorrhage causes blood to effuse from the pharynx.

(3) *Operations involving the opening of one pleural cavity.*—These operations may vary in severity, from the simple drainage of an empyema, to the resection of a large number of ribs or to sternum-splitting for the removal of a tumour. When one side of the normal chest is opened, air enters the pleural cavity, the lung collapses, and dyspnœa immediately occurs. The mediastinum, having no inherent lateral stability, moves towards the unopened side, thus tending to collapse that lung also. It follows that a very considerable muscular effort must be made to draw sufficient air into the sound lung, whilst the lateral movement of the mediastinum always causes a certain amount of shock. If the pleural cavity contains a small amount of fluid, the disturbance in pressure when it is opened will not be so great, while if it is completely full of fluid under pressure, the changes will be reversed. In other words, when such a cavity is opened, the fluid will spurt out, the intrapleural pressure will fall, the lung will tend to expand, and the mediastinum will be displaced towards the opened side. Now the thoracic viscera can adapt themselves to gradual changes in position to a surprising extent. For example, a man may lead a fairly normal life with a tumour occupying the whole of one side of the chest, and with the mediastinum greatly displaced. Sudden changes in position, however, always give rise to a certain amount of shock. For example, it is most dangerous to make, suddenly, a large incision into a pleural cavity which is completely filled with pus under pressure. If the pus is gradually drained off through a wide-bore needle, subsequent proceedings can be carried out with much greater safety.

It has been realized for many years that these undesirable effects arising from pressure-changes can be modified by allowing the patient to breathe air or gases at a pressure higher than that of the atmosphere, and the elaborate pressure cabinets were designed with this object. Later, attempts were made to employ compressed air and ether anæsthesia. These were only partially effective, since the excessive secretions of the respiratory passages necessitated the frequent removal of the mask from the patient's face, thus interrupting the differential pressure. At the present time we have three practicable ways of obtaining positive pressure with anæsthesia: (*a*) The double-airway endotracheal method with a spring-loaded exploratory valve on the return tube. (*b*) The rebreathing intubation method introduced by Dr. Magill and Dr. Rowbotham. The Waters inflatable rubber air-cushion encircling the intubation tube helps materially in obtaining an airtight fit without damaging the vocal cords. (*c*) The third and simplest method is to modify the usual nitrous oxide and oxygen technique. If a continuous-flow apparatus is used, a slightly thicker rubber bag (gauge 8 instead of 10), a spring loaded expiratory valve, and a manometer or pressure-gauge, are desirable. With an intermittent-flow machine no alteration is required to obtain pressures up to 10 mm. Hg, which are usually sufficient.

In my opinion, differential pressure should always be employed in thoracotomies while the pleural cavity is open. The lung should be watched throughout the operation, and the pressure so adjusted that it lies just below the surface of the wound, should this be compatible with the wishes of the surgeon. If this is done, dyspnœa will not occur and post-operative pneumothorax will be avoided. In decortication operations it is necessary to guard against the use of excessive pressure, as the lung cannot be expanded until the thickened pleura has been stripped off.

The position of the patient on the operating table is a matter of some importance in empyema cases, and one in which the views of the surgeon may not coincide with those of the anæsthetist. A satisfactory compromise, however, can generally be arranged. The patient himself is the best judge of what position is permissible. He should be placed ready on the table and if he appears comfortable, anæsthesia may be induced. If, however, the strain of lying on his sound lung gives rise to cyanosis, dyspnœa, or other signs of distress, it is most unwise to begin anæsthetizing until a change in position enables him to breathe comfortably. I am strongly averse to anæsthetizing chest patients in the dorsal position in an anæsthetic room and then letting them be carried to the theatre and turned on their side on the table. It is much safer to settle them comfortably in position before anything is done at all.

(4) *Extensive thoracotomies which may necessitate the simultaneous opening of both pleural cavities.*—The remarks made upon the previous group of operations apply with even greater force to this class of case. If both pleural cavities are widely open at the same time, the most violent respiratory efforts on the patient's part will not cause an adequate amount of air to pass down the trachea, and death from asphyxia will occur unless added pressure is applied. The anæsthetist, therefore, has no choice in the matter; he must use differential pressure. The ways of doing so have already been discussed.

(5) *Intrapulmonary operations.*—Operations involving the division of lung tissue are usually performed with the object of removing a foreign body, such as a bullet, or of draining a pulmonary abscess. In some cases partial and complete lobectomy may be necessary. The lung tissue may be divided by means of the finger or some blunt instrument, by the knife, or by the cautery. In any case a certain amount of blood will be effused into the alveoli and bronchioles and will be coughed up. It is consequently most important that the coughing reflex should not be abolished. If nitrous oxide-oxygen is used, the face-piece should be directed downwards and the blood will then be blown out of the expiratory valve. If the pleural cavity is

opened, differential pressure should be used, but in practice, the presence of adhesions very often renders this unnecessary. If a cautery is used, no ether or ethylene must be employed, as there is a risk of an explosion being caused by the escape of inflammable gases from the ruptured alveoli.

If foreign bodies are removed from the lung on the X-ray table (which is often the case) the anæsthetist's plight is not an enviable one. In addition to the difficulties already described, he has to work in complete darkness for considerable periods on end, and any help which he might obtain from the sound of respiration is nullified by the noise made by the electrical plant. Furthermore, the high-tension leads are usually in unpleasant proximity to the face-piece and a slight movement is sufficient to obtain a severe shock. Finally no inflammable vapours can be used. These formidable difficulties can be mitigated to some extent by having a hooded electric bulb illuminating the mixture control of the gas and oxygen machine, and by feeling the blast from the expiratory valve with the hand, whilst one finger is kept on the temporal pulse. In this way, some idea can be obtained of the respiratory and circulatory condition of the patient, and alterations of mixture can be made. Still, I think that these anæsthetics are among the most difficult that we are called upon to administer.

(6) *Cases in which a bronchial fistula is present.*—Patients who have a large communication between an external wound and a bronchus cannot be anæsthetized with nitrous oxide and oxygen unless the fistulous opening is plugged, since the gases will escape through the wound and a proper regulation of their percentages becomes impossible. The condition is very similar to that of a patient who is breathing partly through his nose and partly through a tracheotomy tube. It is most important never to abolish the coughing reflex if the surgeon contemplates plugging the fistula with gauze soaked in liquid. Cases have occurred in which the excess of fluid has exuded into the main bronchus and has been aspirated into the opposite lung with either fatal results at the time from asphyxia, or the subsequent development of septic broncho-pneumonia. Finally it should be remembered that any cauterization of a fistulous track precludes the use of ether or ethylene.

(7) *Cases in which some obstruction to the respiratory passages is present.*—This obstruction is usually caused by some type of tumour, such as a retrosternal thyroid, dermoid, or sarcoma, pressing upon the trachea or bronchi. The exact position of the obstruction can frequently be located from a radiogram, and if this is possible, it aids the anæsthetist to a considerable extent. In this class of case the necessity for maintaining a clear airway outweighs every other consideration. In my opinion the following procedure should be adopted. The patient should first of all be placed in the position in which he feels most comfortable; in many cases this will be with the head held well forward, so that the tumour lies further inside the thorax instead of being pulled towards the neck where it might compress the trachea between the convexity of the spine behind and the sternum in front. Anæsthesia is then carefully induced with open chloroform until complete muscular relaxation is present. If possible, two stiff catheters are then pushed down the trachea past the obstruction, anæsthesia being continued with endotracheal nitrous oxide, oxygen and ether. In the rare event of one bronchus being partially blocked, it may be impossible to pass a bicoudé catheter down the affected side and thus overcome the obstruction. If this catheter is a small one and if only one is used, both lungs will function. In certain cases of large intrathoracic tumours, it may be possible to extend the head sufficiently to use a laryngoscope; in this event an attempt should be made to pass the catheters through the nose by touch; if this also fails, open chloroform should be continued until the splitting of the sternum or the resection of ribs has relieved the intrathoracic pressure, when the catheters can nearly always be passed with ease. During the subsequent course of the operation it is probable that one or

both pleural cavities will be opened, in which case the factors already considered must be borne in mind and dealt with as circumstances arise.

Several varieties of operations which are, strictly speaking, thoracic in character, have been omitted for various reasons. Manipulations performed through the bronchoscope and œsophagoscope are usually considered under the heading of throat surgery, whilst diaphragmatic herniæ are generally approached from the abdomen.

Cardiac operations are uncommon and, in this country, are practically confined to the removal of foreign bodies. The few cases which I have seen have done well with nitrous oxide-oxygen anæsthesia. I have had no experience at all with valvulotomies for stenosis.

Dr. I. W. Magill: The progress of anæsthesia has done much to facilitate the work of surgeons and extend the scope of operations in thoracic disease.

With efficient methods of anæsthesia at our command we have now the assurance that both pleural cavities can be opened simultaneously, without the risk of immediate death. We also know that the patient's air-way can be absolutely controlled and pulmonary ventilation effectively maintained under most conditions. The knowledge of these facts will undoubtedly influence the surgeon—at least from one aspect—in his decision as to operative risk in thoracic disease, it being now recognized that the management of the anæsthetic in major thoracic operations is of considerable importance.

At the same time it must be remembered that thoracotomy for empyema is a routine operation in every hospital, and operations of this kind have doubtless been performed successfully on many occasions without special anæsthetic skill or special apparatus. Further, the experience of surgeons in the late war has shown that considerable trauma, both operative and otherwise, can be inflicted on the lungs without incurring fatal results and, in the case of operation, without the protection of positive pressure anæsthesia.

In view of the advancing scope of thoracic surgery, it is necessary that we, as anæsthetists, should take stock of the agents and methods at our disposal, and set down the indications for them in the light of our experience up to the present time.

Classification of operations from the anæsthetic standpoint.—Classification of the various pathological conditions requiring operation is entirely a matter for the surgeon, and I do not propose to discuss such simple procedures as drainage of an empyema. It will be agreed that local anæsthesia, supplemented, if necessary, by nitrous oxide and oxygen, is all that is required in these cases.

From the anæsthetic standpoint the major thoracic operations fall broadly into two groups.

(1) Operations which are more or less superficial, such as thoracoplasty, decortication and the treatment of bronchial fistula. I include also the drainage of pulmonary abscess and simple exploratory thoracotomy. The patients in this group are usually in poor condition.

They are subject to frequent coughing to clear the lungs of much secretion. They are usually toxæmic and therefore not good subjects for toxic anæsthetics. In many of these patients the visceral pleura is partially or completely adherent to the chest wall and the operation need cause no respiratory embarrassment at all. In this group, then, positive pressure anæsthesia is not absolutely essential for pulmonary ventilation.

(2) The operations in this group include the removal of mediastinal and intrapulmonary tumours and cysts, operation for diaphragmatic hernia, and excision of the œsophagus and of some retrosternal goitres. In operable cases of this type, in contrast to those in the first group, bronchial secretion may be negligible, and dyspnœa may be absent when the patient is quiescent, even in the presence of a

large tumour; in other words, symptoms may be slight. The method of approach usually entails the opening of one pleural cavity at least, and, as the pleura is usually quite free, pneumothorax is complete. The anæsthetist must be prepared for the accidental opening of the other pleural cavity, but I have only seen this occur once. The removal of the growth is frequently accompanied by much traction on the important structures in the mediastinum, and shock may be rapid and of severe degree. In addition, there is the possibility of hæmorrhage from the large vessels, or the opening of a bronchus during the course of the operation.

It is obvious in operations of this type that the patient requires every possible protection. Pneumothorax is usually induced, where possible, some time before the operation in the course of diagnosis and enables the patient to adjust himself to the condition. This, in my opinion, is of great importance, as the sudden occurrence of pneumothorax in the early stages of a severe operation is liable to be a heavy tax on the resistance of the patient. When pneumothorax is established beforehand, the opening of the thorax at the operation causes no shock or further alteration in the respiratory mechanism. In many of the cases in this group positive pressure anæsthesia is essential for the safety of the patient during the operation. There are certain cases which fall between these two groups. One of these is lobectomy, and in operations of this type, as will be seen later, it is necessary to change from one method of anæsthesia to another when occasion arises.

The anæsthetic.—The anæsthetics at our disposal are local and general, and as in any other branch of surgery, for the minor operations such as resection of one or two ribs, local anæsthesia should be chosen, except perhaps in young children. Local anæsthesia can also be employed for extensive operations such as thoracoplasty, and it is unnecessary to dwell on its advantages. It has been tried at the Brompton Hospital, however, and on the authority of Mr. Tudor Edwards, I can state that general anæsthesia combined with intercostal nerve-block gives better results. When we consider the condition of the patients in group I, it is at once obvious that the general anæsthetic chosen should be non-irritating and non-toxic, and should permit of rapid recovery with an active cough reflex throughout the operation. Fortunately the muscular relaxation demanded in abdominal surgery is unnecessary in these operations, so that ether and chloroform can be dispensed with in most cases, and nitrous oxide-oxygen employed.

This anæsthetic is my own choice for such operations, and I consider that face-piece administration, as opposed to intubation, is all that is necessary, in spite of the difficult position of the patient. Nitrous oxide-oxygen anæsthesia has also the advantage that effective positive pressure can be brought into play when necessary.

It is in the second group of cases that endotracheal insufflation is of the greatest value. Continuous high-pressure insufflation as described by Meltzer and Auer, is unnecessary for most thoracic operations. One should be prepared, of course, to increase the intrapulmonary pressure at once in case of emergency, such as the occurrence of double pneumothorax, or intrabronchial hæmorrhage. I prefer to insufflate at as low a pressure as is sufficient to abolish the dead space in the trachea, and to control expiration directly. This can be done by means of a wide-bore return tube, or by using only one wide-bore tube and an adjustable expiratory valve. Nitrous oxide-oxygen with a minimum of ether is the combination of anæsthetics I prefer, and in these operations it is better to use a minimum of ether than to try to obtain pure nitrous oxide anæsthesia. There is usually no secretion to be expelled immediately after the operation, as in tuberculosis or bronchiectasis, and as there is often considerable shock, immediate recovery from the anæsthetic is not advantageous.

I have endeavoured to classify thoracic operations somewhat broadly into two groups, according to the condition of the patient and the nature of the operation, and I have given lobectomy as an example of an operation which lies between the two. That is to say, patients in whose cases lobectomy is indicated, may exhibit

many of the symptoms described in the first group, such as toxæmia and profuse expectoration; at the same time, the operation entails the possibility of traction on the contents of the mediastinum and interference with respiration, as in the second group. When secretion is marked, lobectomy is best performed under gas and oxygen from a face-piece, positive pressure and, if necessary, intubation, being in readiness should they be required. When secretion is not marked, endotracheal anæsthesia can be employed.

One case of lobectomy is of interest. A boy aged 13 was anæsthetized with gas and oxygen in the ordinary way, and the course of the operation went smoothly until near the end, when there was complete respiratory obstruction. The glottis was seen to be open, and a catheter was passed gently into the trachea until the air-way was re-established, the patient being tilted head downwards at the same time. On application of positive pressure through the catheter, a large clot was expelled from the trachea. This patient made an uninterrupted recovery.

A similar obstruction took place in a young girl about to undergo thoracotomy for the evacuation of pus. In this case the gas mask suddenly filled with pus before the operation had begun. The patient was rapidly placed head downwards, and as the cough reflex was present, this, aided by gravity, expelled about half a pint of pus from the trachea and bronchi. The operation was postponed, and ultimately became unnecessary, as the patient made a good recovery.

An example of the superiority of simple gas-oxygen anæsthesia over endotracheal insufflation is provided by the case of a man aged 27, from whom an intrapulmonary teratoma was removed in three stages. For the first two operations, endotracheal insufflation was employed, using nitrous oxide-oxygen with some ether. The early onset of shock necessitated postponement of removal of the tumour. Both these operations were followed by bronchitis, and convalescence was slow. At the third operation the tumour was removed under gas-oxygen without intubation, and the condition of the patient was markedly better both during and after the operation.

Conclusions.—These may be summarized as follows: (1) Local anæsthesia for minor operations. (2) Nitrous oxide-oxygen combined with intercostal nerve block for major operations in which the pleura is adherent, or not to be opened. (3) Endotracheal insufflation for certain cases only, e.g., when traction on the mediastinum is likely to interfere with efficient pulmonary ventilation.

The anæsthetist should be prepared to intubate at a second's notice, but the wider his experience, the less frequently does the necessity for this arise.

Statistics.—The following figures may be of some interest, and as we are chiefly concerned with the anæsthetic, I have made the classification as simple as possible.

Operation	Number	Deaths (within 6 hours)
Thoracoplasty, Apicolysis, Bronchial fistula, etc.	313	1
Thoracotomy: Exploratory, Abscess, Foreign body	162	2
Operable tumours removed including 7 goitres	29	1
Diaphragmatic hernia	3	—
Excision of œsophagus	3	—
Lobectomy	4	—
	514	4

It will be noted that there were four deaths out of 514 anæsthesias, and these were not strictly anæsthetic deaths. One occurred from collapse following profuse hæmorrhage in a man undergoing decortication. He had been anæsthetized by me on two previous occasions without any ill-effects.

One death occurred from hæmorrhage from a large vessel, in a case of pulmonary abscess; the third from collapse six hours after exploratory thoracotomy for tumour; and the last from collapse four hours after removal of a fibroma weighing 10 lb.

Mr. Tudor Edwards: All recent advances in surgery of the chest owe a great deal to improvements in anæsthesia. The whole question of anæsthesia in thoracic operations depends upon whether operation is carried out with a free pleura or whether the pleura is unopened or adherent. In the former case, some method of positive pressure anæsthesia is essential, whereas in the latter, when general anæsthesia is used, gas-oxygen is the anæsthetic of choice.

There does not appear to be any need, when intratracheal anæsthesia is employed, to maintain more than a minimum positive pressure to permit of inflation of the normal lung, and it is never necessary to increase the pressure sufficiently to distend the lung on the side of operation.

Intratracheal anæsthesia is essential when there is any risk of opening both pleuræ.

Distinct differences exist in the anæsthesia required during thoracoplasties for bronchiectasis and tuberculosis, and those given for chronic empyema. In the former case, the chest-wall mobilization has a direct effect upon the underlying soft lung, whereas in cases of chronic empyema, such a dense fibrous barrier has formed over the lung surface that when the cavity is opened there is no interference with the mechanism of respiration.

Mr. Frankis Evans said that he had had an illustrative case showing the advantage of gas-oxygen over chloroform. On the first occasion the patient was anæsthetized with gas-oxygen, and the after-effects were negligible. On the second occasion, although he was better able to stand an operation (removal of ribs for chronic empyema), he was sick for three days after a very small amount of chloroform. At this second operation, performed by a different surgeon, chloroform had been requested by the operator, with most unpleasant results to the patient.

He (the speaker) did not wish to give the impression that he disapproved of this drug, because he thought it was a most suitable anæsthetic for obstructive conditions, and he took every opportunity of teaching the student its use.

With regard to free-flow syringes, he had found it a simple matter to squeeze the delivery tube while withdrawing the piston, so as to be sure that the needle was not in a vein.

Dr. Z. Mennell said that anæsthetics for chest operations were divided sharply into two classes: (1) When the opening of the pleura caused a free pneumothorax and collapse of the lung; (2) when the pleura was adherent and only a partial pneumothorax was caused.

In the first class, which included such operations as those for exploration of the mediastinum, removal of œsophageal growths or intrathoracic goitres, or for the formidable attempts at removal of clot from the pulmonary artery, then surely a positive pressure in the chest was almost essential. Intratracheal ether in such cases was best for the patient and of great comfort to the surgeon and anæsthetist. In the second class, the patients were often in a serious and debilitated condition and a very light anæsthesia short of muscular relaxation was all that was necessary: this could be best procured by nitrous oxide and oxygen following a preliminary narcotic. Speed on the part of the surgeon was more necessary in these cases than in almost any other class of surgery.

In 1918 and 1919 he had had a series of seventeen cases of Wilms' operation. In two of these, intratracheal ether had been employed, in five, chloroform, and in

fifteen, gas-oxygen (two operations in some cases). They were all "last hope" cases of hæmoptysis or advanced tuberculosis. In three cases death had occurred shortly after operation: two of these deaths (one from pneumonia and the other preceded by an acute delirious condition) were in the cases in which intratracheal ether had been given. The third death was in one of the gas cases, from hæmoptysis. This series had given him the impression that nitrous oxide-oxygen was the anæsthetic of choice.

Finally, he would put in a plea for the use of a light chloroform anæsthesia for many cases of empyema, especially in private houses when the rib was often removed while the patient was in bed. Nitrous oxide-oxygen anæsthesia combined with local novocaine would be ideal, but the ideas of many surgeons in this respect were very crude, and the all-important time factor must be remembered.

Section of Laryngology.

[*February* 7, 1930.]

Demonstration of Apparatus used to treat Functional Aphonia and Allied Disorders, with some Notes on the Classification of such Disorders.

By E. C. MacLeod (introduced by Mr. V. E. Negus).

As a result of the observation and treatment of about seventy-five cases of loss of voice, in the Orthophonic Department, King's College Hospital, it has been found advisable to divide the cases into two main groups and several subdivisions.

(*A*) *True Aphonia*—in which there is little or no approximation of the vocal cords on attempted phonation; the glottis is in the position of normal expiration, or very slightly narrowed. In most cases the cords move normally on coughing, in some male patients, however, the cords close on "fore-arm pressure," but this is not often the case in female patients. The majority of cases of true aphonia are in females.

(*B*) *Hyperphonia*—when the cords are brought forcibly together, and the ventricular bands are also approximated, making phonation almost, if not entirely, impossible. If voice is produced at all, it is either high in pitch and "hard," or very hoarse. Occasionally, double voice is produced, the vocal cord note being relatively high in pitch, with a superimposed note from the ventricular bands, of a much lower pitch.

Groups *A* and *B* are subdivided as follows:

(*A*) (1) Hysterical incoördination: Neurotic condition: organic cause (tuberculosis) excluded. (2) Mechanical interference: Inflammation of larynx—new growth, such as simple papilloma, keeping the vocal cords apart. (3) Muscular weakness: Tuberculosis—after various exhausting illnesses—following prolonged hyperphonia.

(*B*) (1) Hysterical overaction. Primary—following aphonia. (2) Result of neighbouring inflammation. Inflammation of pharynx—after whooping-cough, etc. (3) Resulting from surgical operation: Following removal of papilloma—following laryngofissure—and partial laryngectomy. (4) Misuse: Result of speaking against noise—faulty breath control (too little breath)—over emphasis—faulty training in voice production (singing)—emotional disturbance. Patients in this group are usually teachers, preachers, orators, actors, singers, drill-sergeants, auctioneers, costers, exchange clerks, etc.

The method of treatment which has been found most satisfactory for Group *A* cases is the administration of the faradic current by means of the synchronized stimulator, together with strong "suggestion and persuasion." The apparatus is specially designed to enable these two forms of treatment to be combined, and has many advantages over the sudden, strong electric shock method, and over manipulation of the laryngeal mirror or probe.

Group *B* can best be treated by vocal and muscular exercises designed to relax the existing tension, and to induce gentle phonation. The use of the synchronized stimulator is contra-indicated in Group *B* cases, except on rare occasions.

Discussion.—Dr. Dan McKenzie (President) said that the ordinary treatment by the faradic current, in many if not most cases of functional aphonia, was almost brutal. They had all felt unhappy in applying the faradic current with anything like full force, and if the apparatus shown by Miss MacLeod achieved the result without such distress, they should be very grateful.

Miss MACLEOD explained that there was no particularly unpleasant sensation from the current as applied by means of the apparatus she had demonstrated. The current was a very gentle one, and it was frequently unnecessary to get beyond the stage of "pins and needles." It was pleasant to be able to assure the patient from the beginning that nothing sudden was going to happen, particularly if he had had experience of previous treatment by violent means. The patients had no hesitation whatever in coming back for further treatment if they felt the need of it.

Mr. V. E. NEGUS said it had always seemed to him that the treatment of speech defect should be in the hands of someone interested solely in that particular work. The Orthophonic Department at King's College Hospital was in close collaboration with the Department of Laryngology. The laryngologists saw all cases of aphonia. Organic causes, such as tuberculosis, were first of all excluded, and the others were sent to Miss MacLeod. He had not yet seen a patient with functional aphonia who did not respond on the first occasion. A few minutes after Miss MacLeod had finished with them the patients were usually speaking normally. The cure was generally lasting, but occasionally the trouble recurred, and then they came back again. He had no recollection of one who did not respond the first time.

Sir J. DUNDAS-GRANT said that various procedures had been tried in attempting to restore the voice in cases of functional aphonia. There seemed no limit to the ingenuity that had to be exercised in one way or another, such, for example, as teaching the patient to begin with an inspiratory voice first, and in various other ways. The apparatus shown by Miss MacLeod was certainly ingenious and impressive, but it was not what might be called a "waistcoat pocket edition," and one would like to hear that the results were much better than those which could be obtained by simpler means.

Mr. V. E. NEGUS said that Miss MacLeod was quite aware of the uses of "fore-arm pressure," and found it valuable for some patients, in those for example who had had laryngofissure. This procedure, however, was chiefly useful in men, as could be seen on watching the larynx of a male patient and getting him to exert an arm effort. His experience was that women had not sufficient power in the arms to require to fix the thorax to the same extent on muscular effort; therefore it was of much less use to get women to execute these adduction efforts of the arms than it was in the case of men. As these cases of functional aphonia occurred chiefly in women, not much use for the method had been found at King's College Hospital.

Sir J. DUNDAS-GRANT said that he had found the fore-arm pressure method in the case of women (founded on Mr. Negus's views on the function of the vocal cords in climbing animals) useful, more especially for vocalists in whom fatigue or overwork had resulted in hoarseness and loss of voice, with imperfect approximation of the vocal cords, especially at the posterior part. It was a rather common habit of singers on the platform to place their hands together almost in the attitude of supplication. This need not be altogether a pose on their part; they might in reality be making use of muscular pressure which helped them to approximate their vocal cords and bring out their voice.

Miss MACLEOD, in reply, said that an objection to fore-arm pressure in a certain type of aphonia was that it necessitated general muscular effort. To restore perfect phonation of the voice, one did not want general muscular effort. The procedure certainly was useful in some functional aphonia cases at the outset, but there was a certain danger in proceeding with it, if it did not work at once, for there would be force behind it, and such force would produce a hyperphonic variety of voice. She preferred gentle phonation rather than the phonation which would result from strong muscular pressure applied to close the cords. The fore-arm pressure method was valuable in cases of laryngofissure, in which an extra effort was required to make up the deficiency by increased movement.

Growth on Right Side of Larynx.—H. BELL TAWSE, F.R.C.S.

W. S., male, aged 44. In April, 1915, was "gassed" in France. Aphonia developed, followed by hoarseness, but this lasted only a few days, and he was quite well till early in 1928, when he again became hoarse. The hoarseness increased and dyspnœa developed.

May 15, 1929.—On examination, a fleshy-looking mass, with whitish patches, involved the right vocal cord, ventricular band and arytenoid, and spread across into

the anterior commissure and downwards into the subglottic space. The right side of the larynx appeared to be fixed. Marked dyspnœa. Wassermann reaction negative. No tubercle bacilli in sputum. Chest negative.

May 18, 1929.—Tracheotomy on account of urgent dyspnœa. Diagnosis: Malignant disease of larynx.

May 29, 1929.—Häslinger's directoscopy. Extent of growth confirmed. Pieces removed. Wassermann reaction again negative.

Pathological reports:—(1) *Royal College of Surgeons' Museum, Edinburgh.*

May 30, 1929.—"All the material was sectioned. One of the portions showed a papillomatous condition with some hyperkeratosis. There is a well-marked subacute inflammatory cell reaction in the corium with special abundance of plasma cells which might suggest a gummatous affection, but there is no evidence whatever of tubercle and, if you can trust the Wassermann being definitely negative, there is no sufficient reason to conclude that it is syphilis. There is no evidence of malignancy. The appearance is really that of a small papilloma with a chronic inflammatory sub-epithelial reaction."

(2) *Pathological Department, Nottingham General Hospital.*

May 31, 1929.—"Sections show thickened and keratinized squamous epithelium. There is no infiltration or other sign of malignancy."

In view of these reports, operation was postponed. The effect of a prolonged course of potassium iodide and mercury was negative.

December 4, 1929.—Growth *in statu quo*. Wassermann reaction negative.

Suggestions are requested as to diagnosis and treatment.

The patient is claiming a war pension on the grounds that his present condition is due to "gassing." Is one justified in even partially attributing his disability to "gas"?

Discussion.—Dr. JOBSON HORNE said that as laryngologist to the Ministry of Pensions he had met with similar cases. He would have had no hesitation in attributing the growth in the larynx to being "gassed" and in recommending the man for a pension.

Mr. HERBERT TILLEY exhibited on the screen a drawing of a papilloma removed from a patient whom he had shown to the Section ten years previously.[1] The case had been one of mustard gas poisoning, the patient having been gassed in 1918, and practically from the moment of the gassing, difficulty in breathing had begun, and eighteen months afterwards had become extreme. The lower portion of the trachea was filled with a pale reddish growth which was removed on the day after he (the speaker) first saw the patient, and a section was examined by the late Professor Shattock. He had asked Professor Shattock whether he thought that the mustard gas had caused ulceration of the trachea and that the resulting granulations could eventually have become a tumour the size of the growth which had been removed. Professor Shattock thought that this was the most probable explanation, and he showed that this growth was a papillary granuloma. The history was continuous from the time of the gas attack. As Mr. Bell Tawse had raised the question of gas, he (Mr. Tilley) thought it would be interesting to the Section to be reminded of a lesion which had dated from the inhalation of the gas, and which might have resulted from it.

Mr. NORMAN BARNETT said that everything else having been evidently excluded, the probability was that this was a case of the late results of mustard gas poisoning, which was responsible for some very unexpected conditions.

He believed Mr. Bell Tawse's case came under the category of those unfortunate men who did not "go sick" for every little thing that came along, but struggled to do their jobs, and so had no entry on their Field Service Cards. Other men with no worse gas poisoning "went sick," and in due course got their pensions.

[1] *Proceedings*, 1919, xiii (Sect. Laryng. 3); *Journ. Laryng. and Otol.*, 1920 (Special January Number).

Mr. A. D. SHARP said that any degree of gas poisoning resulting in disability would certainly have been reported in the records of the Field Ambulance. When men were exposed to gas to any serious extent they were unable to carry on their duties for a few weeks—even in the most favourable cases.

Mr. E. WATSON-WILLIAMS said that the question of the date at which mustard gas was introduced should not invalidate the man's claim to a pension. If mustard gas produced such results, he saw no reason why chlorine should not also do so. Those who saw the early chlorine gas casualties in the spring of 1915 could imagine almost any degree of destruction of mucous membrane taking place as a result of exposure to chlorine. His own first case of granuloma of the larynx had been in a man gassed in 1917. The patient had been hoarse for a few days, and then apparently for some time all laryngeal symptoms had disappeared. He consulted him (the speaker) in 1923. He had then been complaining for a few months of hoarseness and spasmodic difficulty in breathing. He was found to have a subglottic growth—soft, faintly lobulated, greyish pink—which could be blown up between the vocal cords. On removal this had proved to be a granuloma. An interesting point in connection with this case was the interval of six years between the time of gassing and that of the appearance of severe symptoms. The man's pension, given on account of laryngitis, was stopped after his clinical examination, when the condition had been diagnosed as a fibroma of the larynx, but a section was sent up with a further claim after operation and the pension was restored. The appearance of the growth was not like that in this case; it was softer and rather smoother and more like that in Mr. Capps' case (see p. 33).

Mr. NORMAN PATTERSON said that had this not been camouflaged in the report as a case of gas poisoning it would have had the label "malignancy." It was difficult of course to get away from the pathological findings.

Mr. W. J. HARRISON had seen two cases of growth in the larynx in pensioners who had been gassed. These also had resembled Mr. Capps' case rather than the one under discussion. In April, 1915, when Mr. Bell Tawse's patient was gassed, chlorine and lachrymatory gas contained in a certain type of field-gun shell were the only gases used. Mustard gas was introduced a number of months later.

Mr. O. POPPER said that before syphilis could be definitely eliminated, a provocative injection of novarsenobillon should be given and the Wassermann test made again.

Mr. J. S. FRASER said he wondered whether this patient would submit to treatment. If he would not do so, the refusal would weigh against his claim. The connection between the condition as seen at present in the larynx and the gassing nearly fifteen years ago was, to say the least of it, rather tenuous.

Dermoid Cyst attached to the Epiglottis.—DAN MCKENZIE, M.D.

Patient, male, aged 28, had noticed a swelling in the throat for eighteen months before his doctor (Dr. Donald Todd-White) saw it and diagnosed it as a cyst.

It presented a remarkable appearance. Bright yellow in colour, and about the size of a hen's egg, with vessels ramifying over it from left to right, it lay across the base of the tongue in such a way as to prevent a view of the larynx. Yet it caused very little discomfort, there being no difficulty in swallowing or breathing, except sometimes during sleep, when it would wake the patient by blocking the larynx, but he was always able, by a short, half-coughing, half-retching movement to propel it on to the tongue and so to clear the airway. To the touch its wall was lax, loose and diffluent, as if the sac was not quite full. Its attachments could not be made out, but its range and direction of movement showed it to be anchored somewhere towards the left side.

It was removed under local anæsthesia without difficulty. The cyst-wall was caught up with Tilley's tonsil forceps, and, after the yellow glutinous contents had been evacuated through the tear thereby produced, the laryngeal mirror showed it to be attached to the left side of the lingual surface of the epiglottis, from which it was then severed by scissors.

The microscope (section of the wall on exhibition) shows it to be "an epithelial proliferous growth," and Dr. Scott-Williamson regards it as a dermoid.

As far as I can learn from a hasty run through the available literature, this seems to be the first time a dermoid attached to the epiglottis has been described. But the likelihood is (and with this suggestion Dr. Scott-Williamson is inclined to agree) that it has developed in connection with the thyro-glossal duct.

Discussion.—Dr. P. WATSON-WILLIAMS asked whether Dr. McKenzie had had any reason to suspect the nature of the growth before he had had an opportunity of examining it microscopically, and if so, what were the distinctive appearances of these rare growths in this region.

Dr. MCKENZIE said that the idea of a dermoid had occurred to him when he first saw the case, but, then, some mucous cysts behaved in curious ways. Of course, it was impossible to tell clinically, or from the naked-eye appearances alone, what the cyst was going to be like, until one opened it.

Unilateral Fixation of the Larynx, apparently Congenital. — DAN MCKENZIE, M.D.

Patient, female, aged 21. The entire left side of the larynx is fixed and the left ventricular band appears rather full. Voice stridulous and husky.

History of hoarseness and throat trouble dating from birth, stridor during sleep being noticed shortly after birth, and breast-feeding being impossible on account of dyspnœa. When aged 2 years was examined under an anæsthetic by Sir Robert Woods, of Dublin, who is said to have reported that there was "a slight enlargement of the left side of the throat."

There is a history of some congenital heart trouble, but that has apparently disappeared, as the patient is now free from any cardiac abnormality.

This case also seems to be unique. The laryngeal picture is of course quite different from that of the better known "congenital laryngeal stenosis."

The parents are extremely intelligent and have watched the development of the case during the whole of the patient's life. I have no doubt that the symptoms have existed ever since birth. The birth was a long and delayed one, but it was not instrumental, and was perfectly natural, so that there was probably no reason to attribute the fixation of the larynx to an injury received at birth. Normal laryngeal sensation is present.

Discussion.—Sir J. DUNDAS-GRANT said that the case looked to him as if there had been some severe inflammatory process and a cicatricial change, producing the fixation.

Dr. MCKENZIE said that the idea of cicatricial change did not appeal to him. One did not see a fixation of the larynx on one side, as in this case, resulting from cicatricial contraction. Why, if it was a cicatricial change, should it be unilateral?

Mr. J. S. FRASER thought that there was dislocation forward of the arytænoid cartilage on the cricoid. He had seen a similar case, in Edinburgh, in which the left side of the larynx was fixed, and it was really a purely mechanical condition, probably due to perichondritis of the joint.

Mr. J. F. O'MALLEY said that judging from the appearance, it seemed to him that there must have been some trauma at birth. It had a suggestion, to some extent, of the thickened appearance seen after a healed laryngofissure. It looked to him as if there was some inflammatory thickening.

Primary (Clinical) Tuberculosis of Larynx.—O. POPPER, F.R.C.S.Ed.

Mrs. H., aged 51. First seen May, 1929. Seven months' history of huskiness of voice. No cough, sputum, or night sweats. Gaining weight. Does not smoke. A daughter died from pulmonary tuberculosis two years ago.

On Examination.—A small mass on the left vocal cord, occupying the anterior two-thirds, but not encroaching on the anterior commissure; smooth and nodulated; no ulceration. Some fullness and redness over the corresponding portion of the left ventricular band. Movements normal. Cord fails to approximate its fellow owing to mass. No unusual pallor of rest of larynx. Interarytænoid space clear. Appearance highly suggestive of epithelioma. *Provisional diagnosis.*—Epithelioma or tubercle.

Wassermann reaction negative. Sputum negative for tubercle bacillus (tested four times). X-ray report on chest (Dr. Rigby): Right apex does not light up so well as left. Calcification both upper lobes suggesting healed tuberculosis. No evidence of active disease. Diaphragm moves well. Mediastinum clear.

Report on chest (Dr. G. Wilkie Scott): No evidence of active tubercle. Right apex: Percussion note less resonant, air entry less, breathing "wavy." Suggestive of old rather than of recent trouble.

Sir StClair Thomson agrees that the diagnosis lies between epithelioma and tubercle. Thinks epithelioma more likely on account of negative findings and general appearance. If sections confirm this opinion, this is an ideal case for laryngo-fissure.

May 28, 1929: Direct laryngoscopy. Three specimens removed. Depression left in place where the tumour had been.

Pathologists' Reports.—(1) Typical giant-cell formation of tuberculous infection (Dr. Kilian Clarke). (2) Typical giant-cell formation (Pathological Department St. Mary's Hospital). (3) Typical giant-cell formation plus tubercle bacilli demonstrated in section (Dr. Storer).

Patient was put under silence treatment; only occasional whispering being allowed. Galvano-cautery applied to affected area on three successive occasions.

August 3, 1929: No sign of active trouble in larynx. A course of emulsion of tubercle bacilli was given, commencing with mgm. $\frac{1}{2000000}$ and rising to mgm. $\frac{1}{5000}$ (latter dose repeated ten times). No variation of temperature throughout.

Present Condition.—Apart from scarring, the larynx presents no unusual features.

Discussion.—Mr. BELL TAWSE asked why Mr. Popper called this a case of primary tuberculosis.

The X-ray report suggested that there had been trouble in the apices of both lungs, and Dr. Wilkie Scott's opinion supported this view. He therefore regarded the case as tuberculous disease of the left vocal cord, secondary to pulmonary trouble. He was rather suspicious of the inner aspect of the left arytænoid.

Mr. O. POPPER said that the features atypical of tuberculosis were that the voice was only slightly below normal, and that although such change as there was dated back for eight months, pulmonary signs were wanting and the weight was increasing. Again, the lesion was mainly anterior and there was no ulceration. The close similarity to epithelioma emphasized the importance of a biopsy in all such cases.

Dr. JOBSON HORNE said he considered that the word "primary" ought to be eliminated from the title of the case. A knowledge of the pathogenesis of the disease precluded the possibility of accepting a theory of primary tuberculosis of the larynx.

Mr. POPPER (in reply) said that he had definitely qualified the term "primary" in the title by adding in brackets the word "clinical," and he thought that, in the absence of any active pulmonary lesion, vouched for "clinically" by the thermometer, stethoscope, microscope (repeated sputum examinations) and X-rays, one was bound to regard the tuberculous process in the larynx, now healed, as having been primary "clinically" to that organ. An old healed lesion in the lung was, he believed, found on autopsy in over 80% of cases in which no symptoms had appeared during life. A lymphatic spread from such a lesion would affect adjacent lung tissue and produce the typical clinical picture of active pulmonary tuberculosis by the time it reached the larynx.

Mr. J. F. O'MALLEY referred to a somewhat similar case of his own in which the patient, clinically, had tuberculosis of the larynx, but in which every possible investigation had failed to discover tuberculosis anywhere else. Evidence of old lesions appeared on post-mortem examination.

Atresia of Left Posterior Choana.—A. L. MACLEOD, M.B.

D. W., female, aged 28. Tonsils and adenoids removed in 1913. Still complained of obstruction. Saw a specialist eight years ago; nothing found.

Present Condition.—Atresia of left posterior choana. No airway. Naso-pharynx clear.

Discussion.—Sir J. DUNDAS-GRANT said that the characteristic feature of this case was the starchy collection of mucus in the obstructed nostril, from which the patient must suffer much discomfort. He was sure that Dr. Macleod would obtain a good result by removing the obstruction surgically if at the same time he chiselled away a portion of the posterior part of the vomer.

Dr. DAN MCKENZIE said it had been lately proposed in Paris that surgical diathermy should be used for the removal of the diaphragm.

Dr. P. WATSON-WILLIAMS said he would advise making a free opening and removing the obstruction as far as possible all round the margin, including the posterior part of the septum.

Case of Nasal Sinus Disease involving the Right Orbit, and accompanied by an Unusual Degree of Deformity.—NORMAN PATTERSON, F.R.C.S.

Male, aged 66. Three years ago noticed "swelling of right eye and pain above it." No headaches or nasal catarrh. Saw doctor who removed polypi from right side of nose. Later on, right side of nose probed: discharge: swelling of eye subsided. Two weeks afterwards, sinus developed on inner side of eye, and there was a profuse blood-stained discharge; at this time there was also a discharge from the right side of the nose and down into the throat. Operation later on by Dr. Hugh Mathias at Tenby Hospital. External incision. Three stitches inserted. Discharge ceased for one week. Afterwards swelling became much greater and eye was displaced forwards, outwards, and downwards. Double vision developed. Pain over supra-orbital region persisted.

On Examination.—Patient is a well-developed man. No history of loss of weight. The right eye lies about an inch below and external to its normal position, and it is proptosed. There is a fluctuating swelling extending from the nose to the outer limit of the orbit. The skin can be easily moved over the swelling, but in the region of the sinus it is inflamed. The sinus is situated in the neighbourhood of what was originally the internal canthus, and from it there comes a somewhat copious foul-smelling purulent discharge, which can be increased by pressure over the swelling. There is a fullness on the right side of the nose which passes into the right cheek and lower eyelid. On examining the nose on the right side, a hard, rounded, smooth swelling is seen projecting from the lateral wall. It practically occludes the nasal passage of this side. On the left side there is some polypoid tissue. [Skiagram shown.]

The case is rather an unusual one. One remarkable feature is that the man has vision $\frac{6}{24}$ in the displaced eye.

Discussion.—Mr. E. MUSGRAVE WOODMAN said that he had had a case of a shop assistant who had come to him with his right eye bulging out of his head. His sight was normal, his temperature was normal, and he was free from pain and any other symptom. X-ray examination had revealed nothing. After local treatment of the nose, which was also apparently normal, the whole condition cleared up and the patient returned home without operation, presumably well. A month later he came up again with exactly the same symptom. The right eye was bulging out a quarter of an inch in front of the left eye

Another X-ray examination was made, and showed a slight suggestion of swelling in the ethmoidal region. He (the speaker) performed an external operation, and opened up the frontal sinus, which was normal. At the back of the eye he had found ethmoidal cells full of thin, clear mucus. He drained through the nose and sutured up the outer wound. The patient had recovered perfectly. The eye became normal, its movements returned, and the sight was unimpaired. It had been a curious case and certainly an alarming one.

Mr. RITCHIE RODGER said that he had had a similar case a few months previously. He had seen the patient several years earlier, when there was a slight swelling in the ethmoidal region, and he had then attempted an intranasal operation, but there had been no evacuation of pus. The patient had returned a few months ago with quite a large swelling, displacing the eye downwards. He performed an external operation, and found a much enlarged frontal sinus completely filled with a material like glue which could be lifted up with forceps in one sticky mass. This explained why no result had been obtained from the intranasal operation, the material being too glutinous to be removed by that means.

Associated Paresis of the Internal Tensors and Abductors of the Vocal Cords in a case of Combined Bulbar Paralysis and Progressive Muscular Atrophy.—MICHAEL VLASTO, F.R.C.S.

About five months ago this patient noticed that her voice was getting weaker. A little later on she found that there was some difficulty in swallowing food. The dysphagia was not more marked with solid food. Until the patient's attention was drawn to the matter, she did not complain of weakness of the upper arms; indeed, the very slowness of onset of the condition had made it difficult for her to analyse her symptoms.

The laryngeal picture is interesting in that it shows, in addition to the bilateral abductor weakness, the typical bowing of the vocal cord on the right side, due to paresis of the thyro-arytenoidei.

Dr. Feiling, who kindly examined the patient for me, reported as follows, December 30, 1929:—

There is slight wasting and some fibrillary tremor of the tongue, with deviation of the soft palate to the left. There is definite muscular atrophy of both sterno-mastoids and trapezius muscles. There is wasting of the muscles of the shoulder girdles and upper arms. The feeble musculature is in striking contrast to the well preserved muscles of the lower limbs. I have no doubt that the bulbar symptoms are due to degeneration of the hypoglossal nucleus and the nucleus ambiguus in the medulla. The atrophy of the arms is due to a similar degeneration of the cells in the anterior cornua of the cervical enlargement of the spinal cord. In other words, the condition is the not uncommon combination of bulbar paralysis and progressive muscular atrophy.

The dysphagia had been the most prominent symptom, and it was for this that the patient originally sought relief. The suggestion has been made that the condition might be due to paralysis of the constrictor muscles.

Discussion.—Dr. DAN MCKENZIE said that he felt emboldened to make a confession, namely, that he occasionally missed a fixed cord. This was particularly the case when the affected cord was lying in the middle line, that is in adduction. He had noticed that no mistake was ever made when examining by the direct method.

Dr. JOBSON HORNE said that the difficulty mentioned by the President in deciding which vocal cord moved, or was impaired in movement, could be overcome readily by the simple device of taking the eyes off the vocal cords and fixing them upon the arytænoid cartilages. The excursion of the arytænoids being wider than that of the vocal cords, the affected one was readily detected.

Mr. O. POPPER said he thought that inability to detect the fixation was due to using only one eye. If both eyes were used, the mistake, really due to an optical disability, was not likely to arise.

Mr. NORMAN PATTERSON said that he had great difficulty in his early days at the hospital in Golden Square in detecting a paralysed cord, and had had a special mirror made for the purpose with a black line drawn on the surface which was vertical when the mirror was placed in position. As the line remained stationary, it was easy to observe slight movements of the cords.

Mr. RITCHIE RODGER said he had had a case of dysphagia of central origin, in which œsophagoscopy had been remarkably easy. The instrument fairly "romped" down the upper part of the œsophagus without encountering any difficulty from the constrictors. The patient had had facial paresis five months previously, supposed to be due to hæmorrhage. This had passed off quickly, as also did the dysphagia.

Dr. P. WATSON-WILLIAMS asked whether the paralysis of the internal tensors of the cord preceded or was coincident with that of the abductors.

Mr. VLASTO (in reply) said that there was abductor weakness associated with the typical bowing of the vocal cord on the right side, but he could not say which preceded the other.

Report of Case of Non-opaque Foreign Body in Left Lower Lobe Bronchus.—V. E. NEGUS, M.S.

An infant, aged 1 year and 10 months, playing with a carrot, inhaled a portion into the air passages. Slight cough supervened. Chest examined same day. No definite signs. Next day, almost complete loss of air entry in left lower lobe, impaired air entry left upper lobe.

Bronchoscopy performed by Mr. Hope, October 19, 1929 (Brunning's tube). One piece of carrot seen first in trachea and removed, and on second inspection another piece was found in the left lower lobe bronchus and was removed. *Dimensions*: Larger piece about 15 mm. by 5 mm., smaller piece about 10 mm. by 5 mm.

The child was perfectly well next day.

A skiagram showed non-return valve obstruction of left lower lobe bronchus.

An Operating Reflector.—O. POPPER, F.R.C.S.Ed.

The silvered mirror is immeasurably superior in reflecting efficiency to those of the all-metal sterilizable type, which alter the quality of the light, especially when they become scratched.

This reflector consists of a removable spring wire handle used in conjunction with an ordinary glass mirror. It is sterilized and inserted into a slot provided and can be adjusted to alter the direction of the beam. It occupies very little space and is inexpensive.

Swellings in Larynx attributed to Gas Poisoning.—F. C. W. CAPPS, F.R.C.S.

J. W., a man, aged 54, has been hoarse intermittently for some years past and attributes the condition to his having been gassed during the war. He has been very much worse during the last two or three weeks.

No history of venereal disease or tuberculosis. Teeth all removed. Tonsils small and fibrous. Nasal septum much displaced to left.

The larynx shows redness and œdema of the false cords. The true cords cannot be seen. The glottis is filled with two pedunculated swellings, the left very much larger than the right, both apparently arising from the anterior commissure. Voice and breathing are surprisingly good considering great obstruction present.

Wassermann reaction "doubtful."

Mr. T. B. LAYTON said that this was a most interesting case. His experience was that when there was a suspicion of syphilis, a doubtful Wassermann reaction distinctly supported that suspicion In this case, in which the lesion was suspiciously syphilitic, a plus-minus Wassermann reaction certainly supported that view. He believed that 80% of tertiary syphilitic lesions did not give a positive Wassermann reaction at all. Therefore, a case with a partially positive Wassermann reaction should be looked upon as positive if this fitted in with the clinical appearance.

Syphiloma of Larynx.—F. C. W. CAPPS, F.R.C.S.

Patient, a man, aged 29, came to hospital complaining of hoarseness for six months past and of aphonia in the morning. No cough. No past history of illness. No family history of tuberculosis. Denies venereal disease. On examination, there is some dental caries. Tongue slightly furred, one or two smooth patches in the posterior third. Mucosa of fauces generally slightly reddened and œdematous. Larynx—general slight reddening. Cords move well; slight thickening and warty excrescence at the anterior end of both. In the region of the anterior commissure there is a subglottic nodular whitish swelling which prevents proper apposition of the cords, but is practically hidden on phonation.

Wassermann reaction: + +; Sigma reaction: 56 units.

Section of Epidemiology and State Medicine.

[*February* 28, 1930.]

Further Experience in Forecasting Epidemics of Smallpox, Plague, and Cholera in India, and its Bearing on the Reduction of Cholera.

By Sir LEONARD ROGERS, C.I.E., M.D., F.R.C.P., F.R.S., I.M.S.Ret.

WHEN I received a kind invitation to contribute a further paper to the Section of Epidemiology I thought it would be a good opportunity to analyse the records of smallpox, plague and cholera in India in the years subsequent to those dealt with in my former studies, to see how far my conclusions had been borne out or not by later experience. My earlier investigation had shown that the variations in the monsoon rainfall exercised a great influence on the prevalence of these diseases in the following year, and thus afforded data for attempting a forecast of their behaviour in the future. I have been bold enough to show my belief in the practical value of my hypothesis by working out a forecast of the probable variations in the incidence of the three diseases in India during the current year, based on last year's meteorological data. This was despatched to India a month ago, and only the main points in the forecasts will be mentioned in this communication.

(I) SMALLPOX.

In my former paper [1], based on forty-eight years' records, I showed that the great decline in the seasonal prevalence of smallpox during the south-west monsoon rains from June to October was closely related to the high absolute humidity at that season, and that the greatest smallpox epidemics in India occurred in north-west and central India following a failure of the monsoon rains, and a season of comparatively low absolute humidity. In the more humid areas of north-east India and Madras relatively low absolute humidities in autumn and early winter are followed by increased smallpox in the following season. With some adjustment for the number of susceptibles, a forecast can thus be made of the probable incidence of smallpox.

Smallpox incidence in 1923 *to* 1929.—In none of these years did the death-rate for all India from smallpox exceed materially the 10-year average up to 1926, the latest complete data available. The chief local variations are shown in Table I. In the Assam Valley the only material rise over the 10-year average rate of 0·63 was in 1926 with 1·20 per mille, and this was related as usual to low absolute humidity from October to December, 1925, aggravated by continued low readings in each month from January to May, 1926. In 1928 a moderate excess in the southern Surma Valley area of Assam also followed low absolute humidity from October, 1927, to January, 1928, and again in February to May, 1928.

TABLE I.—ABSOLUTE HUMIDITIES IN RELATION TO YEARS OF HIGH SMALLPOX.

Province	Ten-year average	Year	Rate per mille	Periods of low absolute humidity favourable to high smallpox
Assam Valley	0·63	1926	1·20	Low Oct.-Dec., 1925, and Jan.-May, 1926
Bengal	0·42	1927	0·9	Low Oct.-Nov., 1926, and in Feb.-March, 1927
Bihar-Orissa	0·40	1926	1·0	Low Oct., 1925, to Jan., 1926, and in April, 1926
		1927	1·0	Very low Oct.-Nov., 1926
United Provinces	0·12	—	—	No year of high smallpox or high humidity
Punjab	0·37	1926	0·86	Low Sept., 1925, and very low in June, 1926 Very high smallpox in July, 1926
N.-W. Frontier	0·30	—	—	No year of high smallpox or low humidity except low monsoon humidity in 1929, and forecast of increased smallpox in 1930
Central Provinces	0·18	—	—	No year of high smallpox or low humidity except low monsoon humidity in 1929, and forecast of increased smallpox in 1930
Bombay	0·24	1924	0·58	Low during 1923 monsoon in Deccan
Madras	0·56	—	—	No year of high smallpox or low humidity

In Bengal the only material smallpox rise from the 10-year average of 0·42 per mille was in 1927 with 0·9 per mille deaths. This followed the lowest absolute humidity rate in October-November of 1926, as usual, aided by further low humidity rates in February and March, 1927.

In Bihar and Orissa the 10-year average of 0·40 rose to 1·0 in both 1926 and 1927. The latter rise followed exceptionally low absolute humidity in October-November of 1926, and the excess in 1926 followed moderately low absolute humidity October, 1925, to January, 1926, accentuated by an extremely low reading in April, 1926, followed by the maximum smallpox incidence in the following month.

In the United and Central Provinces no high smallpox incidence and no significant low October-November absolute humidity readings occurred. The 1929 monsoon absolute humidity was low in the Central Provinces, so an increase in smallpox prevalence is to be expected in 1930.

In the Punjab the only year with a material rise in the smallpox death-rate over the 10-year average of 0·37 was to 0·86 in 1926, and the increase of the disease followed an exceptionally low absolute humidity in the critical month of September, 1925, while the mortality was accentuated by a remarkably low reading in June, 1926, immediately followed by more than double the mortality in July of any other of the last two decades.

In the North-west Frontier Province the smallpox rates were all low, with the highest reading only 0·36 against a 10-year average of 0·30. In 1929 the monsoon absolute humidity was, however, well below the average, and this has led me to forecast a probable increase in the rate of 1930, in spite of the incidence having been exceptionally low in the last half of 1929.

In the Bombay Presidency the only year of the series in which the 10-year average rate of 0·24 was much exceeded was 1924 with 0·58 per mille deaths from smallpox following low absolute humidity during the 1923 monsoon in the Deccan, the largest and most populous part of the Presidency. The 1924 monsoon absolute humidity was also low, but the next year's smallpox was only a little above the average, in accordance with the rule that after high prevalence one year, climatic conditions favouring smallpox have comparatively little effect in the next year.

In Madras once more, none of the eight years dealt with showed either low early cold weather absolute humidity or increased smallpox, in accordance with previous experience.

Summary.—Thus, in spite of the comparatively slight departures from the normal of the climatic conditions in India during these years, all the main examples of increased prevalence of smallpox in the different provinces conformed remarkably closely with the rules I had worked out in my former paper.

(II) Plague.

In 1928 [2] I showed that the saturation deficiencies in four seasons, and the temperatures in the hot weather and monsoon months, all influence the annual and seasonal incidence of the disease. The data of four of these are available before the cold weather seasonal rise in the northern plague-infected provinces of Bihar, the United Provinces and the Punjab, and also to a large extent in the Central Provinces, and are thus of value in forecasting the more severe outbreaks. In the remaining plague area of the Bombay and Madras Deccan the annual rise takes place during the monsoon months, owing to the comparatively low temperature prevailing there, so forecasts are less easy, and depend mainly on the temperature and saturation deficiency in the preceding hot weather months. Both low temperatures and low saturation deficiencies are favourable to the survival of the flea carriers and vice versa, and they were represented by − signs in my tables. The opposite conditions were indicated by + signs, and it was shown that a great preponderance of − signs accompanied high plague incidence and vice versa. Thus, in the Punjab table in the seven years of high and increasing plague only one plus

sign appears, and all the other material deviations from the average were minus ones, and that position is nearly reversed in the six years of increasing plague.

Only the years 1925 to 1929 now remain to be dealt with (see Table II), and the plague mortality for all India shows that none of these have been bad years, while 1927 and 1929 show the lowest rates recorded since the pandemic began in India; this indicates a continued decline of the disease, which is liable to be broken by occasional increases in certain areas. The average mortality in the last ten years, of which full data are available for all British-ruled India, namely, up to 1926, was 210,360, or 0·88 per mille, and the highest rate recently was in 1926, with 0·81 per mille, mainly due to a severe outbreak in the Punjab, with 5·28 per mille mortality, against a 10-year average rate of 2·40, and it is noteworthy that all six factors influencing plague, including four occurring in the later months of 1925, were favourable to high plague incidence in 1926. The other years of the series all showed low or average rates in the Punjab, and it is also noteworthy that in the record low plague year of 1927, with only 0·18 per mille, five of the six factors were definitely unfavourable to plague, and the remaining one showed only a minor contrary degree. A study of the monthly incidence of plague brings out the effects of the factors mentioned even more strikingly.

TABLE II.—PLAGUE INCIDENCE IN RELATION TO TEMPERATURE AND SATURATION DEFICIENCY.

Area	Ten-year average	Year	Rate per mille	Temperature and saturation deficiency factors
Punjab	2·40 ...	1926 ...	5·28 ...	All four saturation deficiencies and two temperatures favourable to plague
		1927 ...	0·18 ...	Five factors unfavourable, record low plague
United Provinces ...	0·93 ...	1925 ...	1·26 ...	Five factors favourable, none unfavourable
		1927 ...	1·78 ...	Four factors favourable, none unfavourable
Bihar	0·36 ...	— ..	— ...	No year high plague, and none favourable to it. Low plague 1927-29 with preponderance of adverse factors
Central Provinces ...	0·58 ...	— ...	— ...	All five years with low plague and preponderance of adverse factors
Forecast for 1930.	Low plague in Bihar and Central Provinces, average to slight excess in Punjab and about average in United Provinces.			

In the United Provinces, with a 10-year average plague incidence of 0·93 per mille, 1925 and 1927 showed the higher rates of 1·26 and 1·78 per mille respectively, and the first showed five and the second four of the six factors favourable to increase of plague, while none were unfavourable. In the two years of low prevalence, 1927 and 1929, the majority of the factors were unfavourable to plague incidence, or they showed little variations from the average.

Central Provinces and the Deccan show the same close correlation between these six factors and the prevalence of plague.

Forecast.—The 1929 climatic data of the first four factors were unfavourable to high plague incidence during 1930 in Bihar and in the Central Provinces, slightly favourable in the Punjab and negative in the United Provinces.

The remaining areas of Bengal, Assam and Madras have little or no plague, so do not require consideration, but the above data will suffice to show that the records of the recent years now dealt with confirm in a remarkable manner the conclusions come to in my former paper on plague and climate in India.

(III) CHOLERA.

Cholera is the most important and interesting of the three great epidemic diseases of India, for out of over 5,500,000 victims in the ten years ending with 1926 in British India, 2,730,000 in round numbers were killed by cholera alone. In my comprehensive memoir [3 and 4] on fifty years' cholera data in relation to meteorology, with 21 maps and 13 charts, I showed that no less than 40 out of 41 epidemics had followed deficient monsoon or winter rains, or both. I also showed that the seasonal rise depends on the absolute humidity, and that cold weather

increase of cholera only occurs in areas such as Assam, Bengal and Madras, in which the minimum monthly absolute humidity falls below 0·400, and the increase during March to May, in Bihar, the United Provinces and the Punjab, depends on the rise of the absolute humidity above that critical point successively from east to west in the huge north-eastern endemic area from Assam to the United Provinces, and not on a spread westward from Bengal, as held for a century, except by Planck, Sanitary Commissioner in the United Provinces, who recognized the truth over fifty years ago (1878), although he did not know what the climatic factor was which influenced the recrudescence of cholera.

Owing to the succession of favourable monsoons, the cholera mortality in British India for the years 1923 to 1929 inclusive, now to be dealt with, was below the above-mentioned average except for a slight excess in 1927, when the total deaths rose to 302,670. Although then there was no very widespread cholera epidemic in these years, local epidemic prevalence occurred occasionally, so it will be of interest to see how far these might have been or were forecast by a study of the antecedent climatic conditions on the lines I described in 1925 [3], and more fully in 1928 [4] (see Table III).

TABLE III.—HIGH CHOLERA YEARS AND RAINFALL DEFICIENCIES.

Area	Ten-year average	Year	Rate per mille	Rainfall deficiencies
Assam Valley ...	1·85	—	—	No year with failure of rains or high cholera
,, Surma Valley	2·29	1924	3·62	Nov., 1923, to May, 1924, minus 16·85 inches rain
Bengal	1·39	1928	1·98	1927 monsoon and winter rains deficient
Orissa	2·16	—	—	No year of low rains or high cholera
Bihar	1·89	1924	3·4	Rainfall deficient Aug., 1923, to May, 1924
		1929	*	1928 monsoon very deficient
Central Provinces	1·21	—	—	No year of low rainfall or high cholera
Bombay ...	0·60	1927	1·36	S. Deccan epidemic after failure of rains
Bombay Sind ...	0·62	1929	1·79	1928 monsoon minus 81% and winter rains failed
Madras	0·96	—	—	No year of low rains or high cholera
United Provinces	0·71	1924	1·48	Excess cholera and low rains in East only
Punjab	0·26	1927	0·55	Epidemic due to spread by Hardwar Kumbh Fair
Forecast for 1930.	Epidemic in East United Provinces and in Bihar in relation to Allahabad Kumbh Fair.			

* Excess cholera, but full figures not available.

TABLE IV.—CHOLERA DEATHS IN BIHAR AND THE UNITED PROVINCES IN ALLAHABAD KUMBH FAIR YEARS.

	Bihar		United Provinces			
Year	Deaths	Per mille	Deaths	Per mille	Total deaths	
1870 ...	3,200*	?	28,441	0·71	31,641	No Fair sanitation
1882 ...	70,458	3·1	89,372	2·02	159,830	,, ,,
1894 ...	119,277	4·9	178,079	3·80	297,356	Good Fair sanitation
1906 ...	107,217	4·4	149,549	3·14	256,766	,, ,,
1918 ...	188,499	7·7	119,746	2·56	308,245	,, ,,

* Death registration only in very limited areas.

ASSAM.

In the Assam Valley the monsoon rains from 1923 to 1928 were all good, and the cholera incidence in 1924 to 1929 was below the 10-year average of 1·85 per mille, except for slight rises to 2·14 in 1924 and 2·16 in 1926, and both coincided with low winter rains from October to April. The Surma Valley 30-year average was 2·29 per mille, and 1927 showed about this rate, and the other years low ones, except in 1924 with the high rate of 3·62. The monsoon rains were good in each year from 1923 to 1929, but the reason for the increased cholera in 1924 was found to be remarkably short winter and spring deficiency of 16·85 inches between November, 1923, to May, 1924, or less than half the normal rate.

In Bengal the average rate is 1·39 per mille, and the only year of the series with a material increase over the average was 1928, with 1·98 per mille, and this was preceded as usual by a deficiency of no less than 16·71 inches of rain in 1927,

or over one-fourth of the total, nearly all in the monsoon and autumn months, aggravated by further low rainfall in January to March, 1928. In 1924 the rainfall in this area was 11 inches short, but this was mostly in the hot season, and there was an actual excess in the critical months of August to November, and little cholera followed.

The Orissa division, including the Puri pilgrimage centre, has had a series of good monsoon rains and has escaped any excess of cholera from 1923 to 1929. The opening of the railway to Puri in 1901 has led to a great reduction in the disease in this, formerly the most cholera stricken division of India, as few pilgrims now march through the district. The disease is, however, spread by rail more rapidly and further than before, especially to the eastern division of the Central Provinces, Bengal, Bihar, the United Provinces and north-east Madras.

Bihar is an important part of the north-eastern endemic area, lying between Bengal and the United Provinces. Its 10-year average cholera incidence is 1·89, and the only year of high incidence in the present series was 3·4 per mille in 1924. This local epidemic followed the high monsoon deficiency of 9·94 inches in August and September, 1923, and the most unusual continued low rainfall from October, 1923, to May, 1924, totalling — 10 inches. In 1928 there was another deficient monsoon with no less than 18 inches below the average, and cholera was very prevalent during the first half of 1929, but the complete figures and rate per mille are not yet available. From 1925 to 1927 there were lesser deficiencies in the monsoon rains, but cholera incidence was low in the two years after the 1924 epidemic, in accordance with the nearly universal law that the disease is never epidemic in the same area in two successive years.

The Central Provinces is an epidemic area, being invaded from surrounding provinces, and may be entirely free from cholera in years of generally low incidence in India as a whole. This was the case in two of the present series of years, namely, 1923 and 1925, and the rate was also low in 1926. In 1927 the cholera rate was 1·17, or close to the 10-year average of 1·21. There was no great failure of the monsoon rains during the series of years under consideration, so that the absence of any epidemic is in accordance with the rules I have laid down for this area.

The Bombay Presidency presents very similar conditions to the Central Provinces. Here, again, in years of generally low cholera in India, the disease may be practically absent from the Bombay Presidency, and it is noteworthy that this was the case in 1925 and in 1926, in spite of the fact that the two corresponding antecedent monsoon rains were rather low on the Bombay Coast.

On the other hand, cholera was epidemic in the Southern Deccan more especially in 1927, with the result that the death-rate per mille for the whole Bombay Presidency rose to 1·36, although in all the remaining years of this series it was well below the 10-year average of 0·60, and this local epidemic presents points of special interest.

The 1927 Deccan Cholera Epidemic Correctly Forecast.

As already mentioned cholera had been practically absent from the whole of the Bombay Presidency during 1925 and 1926. In January and February, 1927, the reported cases numbered 2 and 16 respectively, but there was a sudden rise in March to no less than 2,921, the highest number in that month for fifty years past. The outbreak occurred in the southern Deccan districts, spread rapidly and resulted in a cholera mortality of 4·70 per mille against a 30-year average of 1·67 per mille, while in the Belgaum district the rate was no less than 7·97 per mille. This epidemic followed early termination of the 1926 monsoon rains in September and subsequent failure of the winter rains. The danger was accentuated by the practical absence of the disease in Bombay during the two preceding years and by the prevalence of an exceptionally high absolute humidity at the time of the

outbreak. Every favourable factor for forecasting this epidemic was therefore present, so it is of the greatest interest to be able to report that the divisional health officer, Dr. Pinto, did actually forewarn the authorities of the probability of a cholera epidemic in 1927. Unfortunately it was decided that it was too late to act on his recommendation. It subsequently transpired that the absolute humidity actually rose this year to over the critical point favouring high cholera incidence as early as February, and continued very high in March and April, so, had this factor been known at the time, an almost certain warning of the epidemic could have been given some weeks before the outbreak commenced, and it would have been quite practicable to have prohibited the March Fair in time to stop, or at least greatly mitigate, the subsequent epidemic. A more striking example of the practical value of forecasting cholera outbreaks on the lines I have advocated could scarcely be conceived.

The 1929 Sind Epidemic.

The only other regional epidemic in the Bombay Presidency during this series of years took place in the northern Sind division, where they are rare owing to its being far from the three epidemic cholera areas of India. Only the incomplete weekly mortality returns are yet available, but they indicate a cholera mortality in Sind of 1·79 per mille, against a 30-year average rate of 0·62. Here again, the 1928 monsoon rains were only one-sixth of the normal at Karachi and 81% in defect for all Sind, but practically no rain fell in the first six months of 1929, when the rainfall is always very light, and the 1929 monsoon was a month late in beginning, all these factors aggravating the effect of the failure of the monsoon rains.

The Bombay Presidency, then, has furnished two remarkable examples of the feasibility of forecasting even localized epidemics by a study of the previous climatic records.

The United Provinces and the Punjab are of especial interest owing to the spread of cholera in this area by the pilgrimages to Allahabad and Hardwar. The only year of the present series in which the cholera rate for the whole of the United Provinces was materially above the 10-year average of 0·71 per mille was 1924 with a rate of 1·48 per mille, and the excess was essentially due to a severe epidemic in the two most easternly divisions (Benares and Corrackpur) which followed short monsoon rains of 1923. It is noteworthy that the disease was not epidemic in the more westernly divisions of the United Provinces, where the rainfall had not been short, for this shows the necessity of examining the meteorological records for the particular divisions in which localized epidemics occur, and not only for whole provinces, if accurate forecasts are to be made.

The Hardwar Kumbh Fair Epidemic of 1927.

In the Punjab, cholera was practically absent in 1923 and 1926 with 0·001 and 0·004 per mille recorded deaths attributed to cholera, because they were years of generally low cholera in India as a whole, and also in the United Provinces, whence the Punjab is nearly always infected. In three more years of this series cholera was below the 10-year average of 0·26 in the Punjab, and the only year with epidemic prevalence was 1927 with a rate of 0·55 per mille. As usual, this outbreak was due to direct importation from the Kumbh Pilgrim Fair at Hardwar in April, 1927. This fair takes place every twelve years and is attended by about a million people in the course of a few weeks.

The outbreak started with 58 cases and 28 deaths, and the disease spread by the pilgrim routes both to the Himalayan Garhwal division of the western United Provinces with over 3,000 deaths, and also over the three nearest divisions of the Punjab with 11,286 deaths. Lt.-Colonel Gill, in his annual sanitary report for this year wrote: "The cause of this outbreak was not obscure. It was the direct result

of the Kumbh Mela held at Hardwar in the month of April. . . . Within a week of the conclusion of the Fair fifteen districts were infected in this manner, and some 100 cases and 48 deaths took place in what may be called the primary foci." Needless to say, the most elaborate sanitary precautions had been taken as usual at the Fair site, but unfortunately, the authorities on the spot, who are in the best position to judge, decided that my suggestion of 1926 and early in 1927 [5] to inoculate the pilgrims in their own districts before they started on their perilous journey to Hardwar Fair was not practicable under the present conditions in India. The same conclusion has recently been come to with regard to the Kumbh Fair at Allahabad in January and February, 1930, with the result recorded in recent telegrams from India of the most serious outbreak since 1882, and that, too, in spite of the most careful sanitary precautions. Some three million persons attend this Fair in the course of a few weeks. The full effects of the outbreak will not be evident until March to June with the rise of the absolute humidity to a level favouring high cholera incidence.

The failure of local sanitation at the fair sites to control the spread of cholera epidemics by the pilgrims.—How serious is the mortality due to epidemics spread by the two United Provinces Kumbh Fair pilgrims, will be evident from a glance at the figures below of the cholera mortality and rates per mille in the affected provinces, the eastern divisions especially of the United Provinces and in the neighbouring Bihar in the case of the Allahabad Fairs, and in the western United Provinces and the Punjab, in the case of the Hardwar, for the 30-year average rates per mille include the kumbh years, and if the only non-kumbh years were compared with the kumbh ones the contrast between the two would be still more striking. It was shown in my memoir that high cholera rates in these areas, as well as in the other provinces of India, in non-kumbh years follow short previous monsoon rains, but one of the three worst epidemics in the United Provinces since 1867 was in the Allahabad Kumbh Fair of 1894, in spite of good antecedent rains ; the only important exception to the rule I have found.

Hitherto reliance has been placed almost entirely on good sanitation at the Fair sites for controlling the epidemics. This is an essential measure, but a study of the Allahabad Kumbh year cholera mortalities in relation to the recorded sanitary measures, shows how little such local efforts can be relied on to check the spread of the disease by the pilgrims. In 1870 the United Provinces cholera deaths only numbered 28,441, although it is recorded that the disease was spread by the pilgrims and no organized sanitary precautions were taken at the Fair site in either 1870 or 1882, as recorded by Planck. The reason of the low 1870 incidence is clearly the fact that 1869 was the worst cholera year in the first twenty years cholera records of the province with 92,503 cholera deaths. The 1882 mortality was also considerably less than in the three subsequent Kumbh Fairs up to 1918, yet Planck himself records that no sanitary precautions were taken beforehand at the site of the Fair. He himself found at least 100 persons dead or dying of cholera on the sands on the great bathing day, January 19, and he expresses his surprise that no immediate spread of the disease took place, although infected pilgrims left by the trains. The most elaborate sanitary precautions, including piped water supplies, were taken beforehand during the next three Kumbh Fairs, yet much more severe outbreaks of cholera resulted than in the two former ones without such precautions, the worst being in 1894. The explanation I found in the absolute humidity readings at the time of the Fairs in January and February, for at the time of its occurrence in 1882 the absolute humidity fortunately was extremely low for the season and unfavourable to cholera spread, but in 1894 the opposite condition obtained, and a terrible epidemic followed in spite of elaborate local sanitary efforts.

A still more remarkable example of the importance of the absolute humidity is the fact that the Allahabad Kumbh Fair does not spread cholera epidemics to the

western divisions of the United Provinces, but only to the eastern ones and Bihar, because it is only in the latter that the absolute humidity at the time of the Fair is high enough to permit of epidemic prevalence of cholera. At the time of the Hardwar Fairs in March and April, with much more favourable absolute humidities, the disease can and does spread in both the western United Provinces and in the Punjab. This factor also explains the comparatively low mortality of 10,107 in the Punjab following the 1891 Hardwar Kumbh Fair, the absolute humidity in that year being exceptionally low. The good result was attributed at the time to the sanitary precautions taken at Hardwar in 1891, but in 1892, in spite of further sanitary efforts, the Fair was followed by the most disastrous outbreak of cholera, which rapidly spread throughout the Punjab. No less than 75,959 persons died of cholera in the Punjab alone, and the disease spread through Afghanistan to Europe and even reached Hamburg and this country. The explanation is to be found in the failure of the 1891 monsoon rains and also of the succeeding winter rains, and in the exceptionally high absolute humidity at the time of the Fair.

The causes of the failure of sanitary measures at the Fair sites are not far to seek. In the first place, it is clearly impossible under the conditions prevailing in India to prevent cholera carriers, or those in the incubation stage of the disease, visiting the Fairs. Secondly, it is part of the religious ceremonies to drink the stagnant water in which the crowds are bathing themselves. Something more than the sanitation of the two square miles or so of the Allahabad pilgrim site on the sands at the junction of the Ganges and the Jumna rivers is clearly necessary to prevent the spread of cholera by the three million pilgrims travelling backwards and forwards through the Bihar and the eastern United Provinces.

Inoculation of pilgrims.—The obvious method of reducing the spread of cholera in this way would be to arrange for the inoculation of the pilgrims, either before they set out on their journeys or at railway stations en route. However, this plan is not yet considered to be feasible by the authorities on whom the chief responsibility rests, so other measures must be considered. Anyone with a knowledge of India is aware that it is likely to take centuries to improve village sanitation to the degree required to prevent cholera outbreaks, so protective inoculation appears to be the only feasible mode of combating the disease, and the possibility of utilizing this effective weapon more extensively in India remains to be considered briefly.

In a pamphlet on "The Conquest of Cholera" the Director of Public Health of Bengal, Dr. C. A. Bentley, records data to show how by mass inoculation cholera has been almost stamped out in Korea by the Japanese, in Cochin China by the French, in the Philippines by the Americans and in the East Indies by the Dutch, so it is disappointing to find that this plan has not been made more use of in the great home of cholera, India. That it is feasible to use mass inoculation in India is clear from Dr. Bentley's success in Bengal; two million people were inoculated against the disease in this province during 1928 alone, and the people now know its value. One example recorded by Dr. Bentley will suffice to convince anyone with an open mind on the subject. On the outbreak of cholera in a large village, at first only the Hindus submitted to being inoculated, with the result that the disease soon ceased among them, but continued among the Mohammedans, who then were inoculated, but did not allow their wives to be protected until they saw that the disease only continued among the Mohammedan women. When the women also were inoculated the plague was stayed. Cholera was also widely prevalent in the United Provinces in 1928, but only some 60,000 inoculations were carried out, or 1 per mille of the population against 43 per mille in Bengal.

Fortunately, my suggestion of 1926 to utilize inoculation of pilgrims in controlling the spread of cholera in India did not fall altogether on stony ground, for the sanitary reports for the year 1927 show that Lt.-Colonel J. A. S. Phillips, I.M.S., succeeded for the first time in getting a large number of the Puri pilgrims inoculated

at Puri, Cuttack and Balasore, through which they mainly travel, with "such a very promising start" that a substantial yearly grant has been made to continue "this very effective measure," and in the Central Provinces, pilgrims returning from Puri were inoculated voluntarily at a railway junction with such success that it was decided to extend the measure to all the important fairs in the province (see Map XVI of my memoir for evidence of the infection of the Central Provinces by pilgrims returning from five different fairs in 1906 alone). Dr. Bentley also had a number of returning Puri pilgrims inoculated on arrival in Calcutta. Once cholera has broken out among pilgrims, there is comparatively little difficulty in getting contacts inoculated against it. This measure, however, savours rather of the policy of closing the stable door after the horse has escaped, and as it is universally the custom to disperse the pilgrims as soon as an outbreak of cholera occurs, there is little time for inoculation, so it cannot be compared in efficacy to my proposal to inoculate the pilgrims before they go to a fair. Nevertheless, the recent adoption of the plan of inoculating pilgrims in Bengal, Bihar and the Central Provinces, is likely, before long, to educate the people up to the point when they will be ready to submit to inoculation before, instead of only after, exposure to infection; for even the uneducated people in India are quick to recognize the value of such a measure, once they have had an opportunity of witnessing its good effects. We may therefore now look forward to the not distant day when the potent weapon of mass inoculation will be used in India, as elsewhere, to the extent necessary to enable a great reduction to be made for the first time in the annual holocaust of victims in India from such a terrible and easily preventable disease as cholera.

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Discussion.—Sir WILLIAM HAMER: Sir Leonard's papers of 1926 and 1930 call to mind those of Dr. Longstaff, read in 1880 and 1905, on meteorological conditions affecting certain epidemic diseases in this country. As regards both England and Wales and India, important conclusions were formulated in the earlier papers, and later the facts were brought up to date and the original results were, broadly speaking, confirmed. The difficulties presented by the problem of disentanglement of co-operating factors were, however, found to be even greater in the later than in the earlier papers. Both investigations recall Sydenham's difficulties of 250 years ago and his conclusions thereon, summarized in the early chapters of his "Medical Observations" and elaborated in Section IV, iv, 3 and 4, in the great first chapter of Section V and in the early paragraphs of the Brady Letter.

Sydenham attached primary importance to interaction between the humours of the body and the "secret temperature of the air," meaning by the latter, the modern pathogenic germ or ultramicroscopic virus. The meteorological conditions (Sydenham's "manifest qualities of the air"), and, perhaps we should now add, other contributory factors (secondary or associated organisms and enzymes) all promote or retard the working of the main factor, modifying symptoms in particular prevalences, but "the fever" (so Sydenham styled it) "remained the same throughout." "By this contrivance," as he phrased it, "Nature manifests her superior power in the production of epidemic diseases."

While recognizing the paramount importance of this discovery by the Founder of Epidemiology, epidemiologists are beginning more and more fully to appreciate the significance and helpfulness, in suggesting preventive and curative methods, of adjuvant accessory factors, such, for example, as "manifest qualities of the air." Hence the great value of such inquiries as those of Longstaff in England, and of similar inquiries relating to epidemic diseases in India, for which the Section is so much indebted to Sir Leonard Rogers.

Fleet-Surgeon W. E. HOME : The profession has always ascribed much importance in the causation of disease to meteorological conditions, but without definitely explained reasons. When I joined the profession, climate was thought to be the cause of nearly all the diseases met with in hot countries, but against that theory I always protested. It might be untrue ; it encouraged fatalism ; it fettered research and initiative, for to change the climate was beyond our power. What we needed was, and is, a knowledge of the biological occurrences which link meteorological conditions to variations in disease prevalence.

In 1919, Bulgarian prisoners of war were coming home, and S. Konsuloff expected a great outbreak of malaria, but that proved to be really a light malaria year. Konsuloff investigated the meteorological records, and found that the first brood of larvæ were washed out of the pools by fierce rain squalls in March-April, and the next brood six weeks later, so in that year rain actually materially diminished mosquitoes and, consequently, malaria.

As to to-night's paper, dry weather increases cholera by decreasing the water supply, lessening the dilution of the cholera cultures in tanks and pools and streams. But is this the only reason ? At any rate Sir Leonard Rogers' patient and brilliant work has already made some forecasting possible ; who can say how great may be its influence in the future.

Dr. J. D. ROLLESTON asked Sir Leonard Rogers if he knew whether any cases of post-vaccinal encephalitis had been reported among the very large number of vaccinations which had been performed in India. It was a remarkable fact that in those countries in which smallpox was mildest, namely, England and Holland, post-vaccinal encephalitis was most frequent, whereas in those countries in which smallpox assumed a more virulent form, such as Roumania, Czecho-Slovakia and Soviet Russia, no cases of post-vaccinal encephalitis had been reported.[1]

Sir LEONARD ROGERS (in reply) said that the reason why he had been fortunate enough to obtain such definite results from his epidemiological studies was because India presented unique opportunities for the work in the immense population with uniform statistics for a number of decades, and the extremely varying climatic conditions in different parts of the country. In reply to Dr. Rolleston, he had not met with any records of post-vaccinal encephalitis in India, and the 1928 Sanitary Report for the United Provinces stated that none had been met with. It must, however, be remembered that only a very small proportion of vaccinated children were ever seen by a medical man in India, so that cases might easily escape observation.

1 *Bull. Office Internat. d'Hyg. publ.*, 1929, xxi, 1183.

Section of Electro-Therapeutics.

[*January* 17, 1930.]

Cutting Currents.

By JOHN ANDERSON, F.R.C.S.Ed.

FOR just over six years I have been conducting a clinical investigation on the uses of the cutting current in the ordinary work of a general surgeon. The bulk of this work has been done in its application to radical operation for operable cancer of the breast, but this has led to numerous other uses, and I am more than ever convinced, since I recently used the Cushing-Bovie machine, that we are now only on the margin of its field of application. This investigation originated in September, 1923. While on a visit to Stockholm I saw at the Radium Institute, presided over by Dr. Gösta Forssell, radical excision for breast cancer by the arc cutting current, and certain problems came immediately to my mind which appeared to demand trial and investigation. The method seemed to me to be technically sound and to give us an addition to our armamentarium in our attempt to combat dissemination of the cancer cell. As I saw it, however, it seemed to have certain distinct disadvantages. An unnecessarily wide area of skin was excised and a large open granulating wound was left, which took several months to heal. On the other hand, the extent of operative procedure was not so wide as we practise with scalpel excision. I felt sure that in this country, patients would be unwilling to submit to a method entailing such protracted convalescence, and our beds and staff would be taxed in dealing with them. I also felt sure that unless primary union could be obtained, the method would be condemned and cast aside, and the first problem that should be settled was: Can we still use the method and obtain primary union? I was fortunate in the fact that the first instrument placed at my disposal, namely, the Schall apparatus, has proved, until recently, by far the most efficient arc cutting instrument that I have tried. I have since investigated at least ten other instruments, all reputed to cut well, and I have been continually disappointed with their action on living tissue, although frequently the effect appeared quite good on dead flesh. The Cushing-Bovie apparatus, which I tried ten days ago (and of which I will speak later) appears, however, to give a more accurate and efficient cutting current. It certainly has a much wider range of application and I have every reason to believe that the healing will be equally good.

We first tried the method on dead flesh (bovine and human), then went on to excise a simple tumour from the breast, and obtained primary union, and from that straightaway performed a complete radical Halstead operation for breast cancer, except that I dissected the axillary portion, near the vessels and nerves, with the knife. In this case I obtained perfect primary union without drainage. Since then I have used the method in nearly one hundred cases of radical excision of breast cancer, and primary union has been the rule. In only a relatively small number have I found any delay or difficulty in healing. In fact these cases have had a very much shorter stay in hospital than those in which the operation was performed with the knife.

When I came to investigate those cases in which there was delay in union, I could, in almost every case, associate them with some little technical inefficiency in the instrument, owing to which there was more coagulating current and less arc cutting, with consequent delay in the healing process, that is to say, there was more dehydration.

Considerable diversity of opinion still appears to exist among electrical experts as to the exact nature of the various types of currents produced by various diathermy machines, but I think that to-day we understand a little more about how these currents act. The small Schall machine produces an excellent cutting-arc, with an average of a tenth of a millimetre of dehydration, as measured by photomicrographs of the tissue. The frequency is high, probably over a million. It can be used as a coagulating current when the contact is made before the current is turned on. Depth of dehydration can only be regulated by using different electrodes, or by contact current. The heavier the electrode, the deeper the dehydration, and the more rapidly the active electrode is working, the lighter the dehydration.

In the more modern apparatus now at our disposal we shall probably be able, with a little experience, to regulate accurately for the different tissues, the amount of dehydration and the rapidity of cutting, because in this apparatus we have a variable current giving a scale of power, a scale of voltage, and a scale of dehydration. With the small Schall apparatus I have been unable to dissect in the neighbourhood of the larger nerves on account of the jerking and twitching produced. These are probably due to "undertones." Whether these are directly produced from the machine, or whether they are induction currents, it is as yet difficult to say. That induction currents are produced is almost certain, as we have found during errors in our technique by which we got monopolar cutting when the patient and machine were heavily insulated.

THE USES OF THE DIATHERMY CUTTING CURRENT FOR THE GENERAL SURGEON.

Biopsies.—For all biopsies I use a diathermy cutting current. I think it eliminates the risk of manipulative spread of the cancer cells which is always in one's mind when one does an ordinary biopsy with the knife. This has led me to perform biopsy more frequently than I previously did and even in cases in which radium is to be used, we can complete the academic picture without increasing the risk to the patient. When the tumour is large I use circumvallation and then cutting current excision. When it is small, circumvallation is liable to interfere with the histological appearance of the section.

Cancer of the Breast.—I have used the method in almost a hundred cases. I fully realize that no end-results of real value can be given until at least eight years have elapsed, but up to the present we have had only two cases which have shown local recurrence, apart from general carcinomatosis. There has been no case of secondary hæmorrhage and no marked protein shock. There is less post-operative pain and much less post-operative shock (capping of nerves); healing has proved more satisfactory, stay in hospital has been lessened, and recurrence of the disease is, to date, definitely less frequent. In inoperable cancer of the breast one frequently removes an inoperable tumour for the comfort of the patient, and one has been struck with the absence of that lighting-up of the disease which ordinary operative procedure so frequently appears to stimulate.

The Bladder.—I use this method for excision of all tumours of the bladder, and cystoscopic control has proved that healing is particularly good in this area; in fact the suprapubic knife-wound is always more obvious than the site of a diathermy excision.

Prostatectomy.—After the ordinary open operation of Thomson-Walker I have found the diathermy current a great advantage, as compared with suture or ligature, in producing hæmastasis. I also find it useful to "titivate" a prostatic bed. In cases of small fibrous prostate I have used both coagulating and cutting current for partial prostatectomy and to allow the central application of radium. In none of these cases has there been any marked degree of secondary hæmorrhage.

Cancer of the Tongue.—For removal of the tongue we are commencing a series of different types of operation as a control: (1) A barrage of radium needles alone; (2) electro-coagulation of growth combined with a barrage of radium needles; (3) excision by cutting current; (4) excision by electro-coagulation. In all excision cases we perform a primary laryngotomy and thoroughly pack the naso-pharynx. We find that this procedure has diminished our mortality and made the risk of operative procedure almost negligible. The absence of post-operative complications—for example, infective pneumonia, post-operative pain, and hæmorrhage—has been very striking. In the majority of these cases the patients get up on the second or third day and leave hospital within ten days. We now operate by this method as a palliative procedure for the comfort of the patient in many cases of cancer of the tongue which we would not formerly have dared to touch, from the point of view of primary operative risk, occupation of hospital beds, or lengthened convalescence.

We use this method for the majority of skin tumours and for tumours of the lip and mouth. That I have not made the application wider (for example, to the abdomen, and to tumours of brain and lung) has probably been due to two causes: first, my feeling that we cannot afford to experiment until we are relatively certain; and second, that up till now we have not had apparatus available by which we could sufficiently regulate the action of the current on the tissue concerned.

Criticisms.

My colleagues often say: "But it is not a bloodless operation." It is not intended to be a bloodless operation. What we claim is that less than half the amount of blood is lost than is lost in operation by the knife. If we use a coagulating cutting current we can secure a bloodless operation, but we cannot then expect to get *per primam* healing. There are two types of operation to be considered. When we are cutting through relatively safe tissue and simply attempting to prevent manipulative spread of the cancer cell, the arc electrode with its tenth of a millimetre of dehydration is sufficient to prevent dissemination, by sealing all the lymphatic vessels. At the same time this prevents all capillary bleeding. In this case all major vessels are taken with forceps and sealed by electro-coagulation. When it is necessary to cut near the tumour, in what we might call the dangerous area, electro-coagulation contact cutting is to be preferred, as one's first aim must always be to deal with the disease rather than to consider the healing. A considerable number of cases have been reported in which the healing was bad and the edges of the wound showed sloughing. With these cases also I am familiar, and they have always been associated with a machine which is not working properly, or with deficient technique, that is to say, coagulation was too deep and dead tissue was left which the natural process of repair could not overcome.

Why then is this method not in more general use? There remains in this country and probably in others, at least if one is to judge by the literature, considerable uncertainty as to what these currents do: their exact nature, their effect on tissue, the different currents produced by different machines, and how exactly to apply the currents to different types of cases and to different tissues. There appears to be a lack of liaison between the electro-therapeutist, the surgeon, and the physicist or electrical expert who designs and builds the machines. Speaking for the surgeon, and for myself in particular, I find myself still grossly ignorant of many of the electrical problems in surgical diathermy. I find that many surgeons wish to establish the method in their clinics, but they find difficulty in procuring an efficient machine which will give a uniform output. For example, with alternating current, until recently, I have been unable to get efficient results. It appears to me, therefore, that until we get

some real standardization of machines, surgical diathermy will not take the place in this country which efficiency demands. I find that the surgeon is zealous for his surgical material and unwilling to hand it over to the electro-therapeutist for what he considers experimental purposes, with the result that often the surgeon is using an apparatus which does not do his work properly, and the electro-therapeutist is confined in his activities because he has not the clinical material at his disposal. The result is that there is no one to tell the physicist exactly what is required, and the fact remains that in Europe we appear to have produced very few, if any, machines which give a good cutting current. I feel that if real team work could be obtained, some uniformity of apparatus could be assured, and until that is done uniform results are almost impossible.

When we read of Harvey Cushing's successes in removal of unencapsuled brain tumours, we can realize the possibilities of the method.

At the moment there is a remarkable wave of radium research. I think that this is right, and will lead to an exact definition of its potentialities and deficiencies, but the plea I make is that we should not lose all sense of proportion, and should realize that until we have some specific agent for the treatment of the cancer cell, we must use not one, but all the means in our power to prevent its dissemination.

For the investigation of surgical diathermy, its efficiencies and deficiencies, I would suggest the coöperation in research of a physicist, a biochemist, a surgeon, a histologist and an electrical expert. There are all kinds of problems requiring investigation. For example, what is a spark or arc? What is its exact effect on tissues? Does it affect staining reagents by electrical means? What is the effect of the electrical current on local anæsthetics impregnating a wound? Is the process of the cutting current a stream of electrons hurling tissue aside? Is it not possible that the size, shape and position of the indifferent electrode and its distance from the active electrode, may influence in some degree the exact uniformity of results? It seems to me that in the cutting current the type of active electrode influences the angle of incidence from the current more than anything else does. The finest cutting we have obtained was from a fine scalpel blade, the next from a fine needle, the next from a thicker needle of the darning type and the last from the heavy electrode. These electrodes and adjustment of spark-gap were the only methods by which, in a Schall machine, we were able to control our depth of dehydration.

With regard to the examination of histological changes occurring in the cells after the use of the diathermy current, I would like in the near future, to make a series of sections by immediate freezing, instead of by the prolonged hardening method which we have used up till now. It is just possible that in the ordinary process of preparation, certain chemical changes take place in the cells, altering them in appearance, so that it is difficult to say which of these changes are due to the diathermy current and which to the chemical process.

We have, as I have said, recently tested the new Cushing-Bovie machine, which appears to be a long way ahead of anything we have yet tried. It gives efficient cutting, coagulating, and desiccating currents, all of which can be regulated and inter-regulated in power, voltage and dehydration. The dehydration appears to depend almost entirely on the nature of the damping of the wave, a purely undamped wave giving practically the same effect as scalpel cutting, with an infinitesimal layer of dehydration. It is only when the damping element is introduced, in various degrees, that we get the different degrees of dehydration required for the different tissues with which we have to work. We tested the machine on tongue, brain, ordinary muscle, fat, cartilage and bone, and we removed two rodent ulcers of the face and a simple tumour of the breast, and carried out a total excision of the breast and fascia for early papillary growth. The whole of the breast operation was done with low voltage and low dehydration. The maximum power used at any part was 50%,

which was used for sealing vessels. Most of the work was done at 35%. The fat was cut at 30%, fine dissections at 20%, and fine skin marking at 2½%.

Dr. Norman Dott tells me that he is getting efficient cutting current from the new portable Wappler machine, and that with this he is able to vary the current over considerable range. I have not yet been able to confirm this, but I know that Dr. Dott has familiarized himself with this machine while on a recent visit to America.

I find that fine dissection is most accurately carried out by using the electrode as one would do a pencil in sketching.

Observations made during Investigations of the Cushing-Bovie Machine.

Dry Tissue.—Best with low current and low dehydration.

Cartilage.—Cuts well with high voltage. Would not cut well with low voltage.

Tongue.—Same as cartilage.

Note.—When coagulating tissue with the needle and it sticks, change to cutting current, which allows withdrawal. (Tested on brain).

N.B.—Put switch off before changing the current. It appears to volatilize the tissue around the needle.

With Light Dehydration.—By making arc with snare, a portion of brain can be cut out rapidly and finely, leaving a fine layer of coagulation behind.

Note.—In cutting out portions of dead tissue with the loop or snare, about 3 cm. of wire was used, the speed being 1 cm. per second.

Heavy Dehydration.—Was slower and produced more cooking. (*Aside*: Theoretically estimated at one-tenth mm.).

Using Snare on Cartilage.—Best with low voltage, light dehydration and high power. Low power was no good.

Nasal Septum.—Cut readily and easily with low voltage, light dehydration and high power. No cutting with low power.

Bone.—High power with no dehydration, cut bone like cheese.

Fat.—Best to use heavy dehydration.

Operation on Face (*living subject*).—30% of power with low voltage and low dehydration.

Discussion.—Dr. G. B. Batten said that at the present time, unlike the position in the early days of radiology, the knowledge of these high-frequency currents "damped" and "undamped" was, thanks greatly to the development of "wireless," immediately available. It required only the demand to bring forward the knowledge possessed by teams, to design really efficient and perhaps portable diathermic machines. Mr. Anderson's work should quickly create such a demand.

Dr. Albert Eidenow said that during the past six months he had attempted to design a surgical diathermy apparatus. There were many difficulties in the making of these apparatus, since the current used was a resonant current, and it was difficult to obtain a high capacity with high voltage current (i.e., 20-30 k.v.) and high frequencies in the nature of 1-2 million per second. All apparatus should be supplied with a voltmeter and a meter to measure oscillations directly; this was a great necessity, as otherwise it was impossible to determine the value of the present diathermy machines. The electrical measurements of many machines were very vague, and so far, the only guide to their efficiency had been based on clinical trial. No progress could be made with such apparatus until the electrical energy and measurements had been correctly determined.

Mr. E. W. Riches said he had seen Mr. Anderson carry out a radical excision of the breast by this method in 1926, and had been impressed by its many advantages, particularly in the small amount of bleeding and the neat and rapid way in which bleeding points which had to be picked up, could be sealed off. Mr. Anderson had at that time also shown a number of cases of breast amputation performed by the cutting current, and in all of these the scar was sound and supple. The method had subsequently been tried fairly extensively at the Middlesex Hospital by different surgeons, using the same type of machine as in

Dundee. At first a Bard-Parker knife was used as the active electrode, but he (the speaker) was doubtful if this was the best type; it soon became covered with a charred coagulum which had to be scraped off. In answer to Mr. Anderson's question as to why the method was not more in use, he suggested the following reasons: (*a*) There was a certain variability in the working of the machine, as well as a variability in different patients; it was never so good in a fat patient. If the electrode happened to stick in one place, it produced a slough, and that prevented primary union. (*b*) Even in cases in which everything went well, there was some delay in healing; these wounds took about two days longer to heal than those made with a knife. (*c*) If costal cartilage were cut, it underwent necrosis; all surgeons did not realize the necessity of avoiding the costal cartilages. The large machine which had just been demonstrated performed its work well on dead tissues, but smaller and less expensive machines would do the same thing. He wondered whether the specific advantages claimed for this machine justified its increased cost.

Mr. ANDERSON (in reply) said that he had ceased to use the Bard-Parker knife as the active electrode because it was difficult to insulate and did not give so fine a cutting section as the platinum knife. He believed that the charred coagulum mentioned by Mr. Riches was mainly due to a mixed current coming from the machine and not really the fault of the knife itself. There was no doubt at all that the Cushing-Bovie machine constituted a great advance on any machine previously seen in this country, but whether its advantages justified the considerable increase in cost remained to be proved by clinical investigation.

Section of Dermatology.

[*January* 16, 1930.]

CASES.

Erythema Circinatum, ? Erythème Annulaire Centrifuge of Darier.—E. G. GRAHAM LITTLE, M.D.

Patient, female, aged 30. This case has, I am afraid, lost some of its characteristic features. Three weeks ago there was a well-marked circinate erythema with a raised edge, forming a gyrate marginate patch on the forearm. The patient has had the same kind of eruption every year for the last four years. The individual lesions are described as minute rose spots, which enlarge centrifugally to form these figurate patches. The duration of a patch is about four weeks. The diagnosis of "Erythème annulaire centrifuge" is suggested.

Folliculitis Decalvans.—E. G. GRAHAM LITTLE, M.D.

Patient, male, aged 35. There is a definite peri-follicular inflammation in some of the patches, upon which there is now a cicatricial alopecia. There is no lesion elsewhere on the body or in the mouth. The loss of hair was the first event noticed, and the whole series of symptoms is not of longer duration than three months. We have recently seen a similar condition. In some cases, patches of lichen planus are present. The question must be considered whether in many cases the conditions described as folliculitis decalvans are not really varieties of lichen planus, of this unusual type. In those cases, few of which have as yet been recorded, the cicatricial atrophy has usually long preceded the other symptoms. In the first case I showed there was an interval of ten years, and that case developed lichen spinulosus. In a case I showed a short time ago the period was shorter, and the lichen spinulosus preceded the alopecia. I think that folliculitis decalvans is uncommon in men, and the syndrome I have just mentioned appears to be more frequent in women.

Discussion.—Dr. S. E. DORE (President) said he did not remember having seen a case with such a short history; one usually saw them at a stage when there was a better-defined margin and more atrophy. He agreed that the disease was much commoner in women than in men.

Did Dr. Graham Little draw a distinction between folliculitis decalvans and the pseudo-pelade of Brocq, or did he regard one as a further stage of the other?

Dr. GRAHAM LITTLE said that he had always regarded pseudo-pelade as an end-product of more than one disease. One called the condition "folliculitis decalvans" when inflammation around the follicles was present.

Dr. DOUGLAS HEATH said that some of these eruptions were, probably, not true lichen planus; they might be instances of a peculiar lichen scrofulosorum in an adult.

Dr. H. W. BARBER said he agreed with Dr. Graham Little's diagnosis and suggested that, as the condition was in such an early stage, a section should be made. The lesions were definitely pink, whereas in most cases of pseudo-pelade, there was more atrophy, and the patches were white.

With regard to the President's point as to whether pseudo-pelade was the same condition as folliculitis decalvans, he (the speaker) thought there were two groups of cases of so-called pseudo-pelade: one in which there were plane atrophic white patches without evidence of inflammatory reaction at the mouths of the follicles: the other with plane denuded flat patches, but also a hyperkeratosis and an inflammatory halo at the mouths of the follicles round the margins of the patches. When the follicular lesions were present, he suggested that they were the same as lichen spinulosus on the body, whereas the plane patches represented atrophic lichen planus occurring on the scalp.

In answer to Dr. Douglas Heath, he would point out that in a considerable proportion of cases there had been lichen planus lesions on the mucous membranes.

Parapsoriasis.—E. G. GRAHAM LITTLE, M.D.

Patient, female, aged 50, admitted to hospital with pronounced symptoms of tabes. The Wassermann reaction was negative, but I thought at first that the rash might be a syphilide. The character of the eruption has not changed in seven months.

Discussion.—Dr. G. B. DOWLING said he had had a similar case in which, however, the eruption was more widely spread. There had been yellowish patches, not scaly, scattered all over the body, and the prominent symptom had been itching. The patient in the present case also experienced irritation, especially at night. Irritation had been said to exclude the diagnosis of parapsoriasis, but he did not agree with that statement. He had indeed often seen *parapsoriasis en plaques* with a good deal of itching. He regarded this case as an atypical parapsoriasis.

Dr. H. W. BARBER said he agreed with Dr. Dowling that the question of itching should not influence the diagnosis at all. He now had under observation two typical cases of parapsoriasis, and both patients complained, at certain times, of definite irritation. The question of itching was, he thought, one rather of the particular patient than of the eruption.

Dr. A. C. ROXBURGH said he had at the present time a similar case to the one described by Dr. Dowling, the lesions being widely distributed. The lesions in this case were more pigmented, and there were more "cayenne pepper" spots. The patient had at first complained that the lesions itched, but the itching had been subdued by the application of liquor carbonis detergens. The eruption resembled that of Schamberg's disease.

Lichen Planus Bullosa.—E. G. GRAHAM LITTLE, M.D.

This patient, now aged 74, was under the care of Sir Malcolm Morris twenty-seven years ago, on account of an eruption which, she says, was similar to this, and was treated by him for three years, and afterwards she was free from the condition for twenty-five years. Two years ago the eruption re-appeared and she came to me this afternoon for the first time. There are hypertrophic patches of lichen planus on the lower limbs, and a number of flat bullous lesions, interspersed with the lichen planus patches, on the leg: there is a very definite and extensive bullous eruption in the groins and on the labia majora, and the inside of the vulva is occupied by definite lichen planus "leucoplakia." A similar condition is to be seen on the tongue. It is obviously recurrent lichen planus, after a long period of freedom, with a bullous eruption in addition to the commoner lesions of lichen planus.

Discussion.—The PRESIDENT said that bullæ were a very rare complication of lichen planus. He had shown a case at the meeting of the British Association of Dermatology three years ago. The only other case he remembered having seen had been under the care of Sir Malcolm Morris. In that case the patient, a man who had lichen planus, with bullæ on the shins, had taken arsenic.

Dr. G. B. DOWLING said that a week ago he had seen an acute case of lichen planus with the typical eruption on the trunk, and here and there on the limbs there were bullæ, the largest of which was the size of a small egg.

Mycosis Fungoides.—H. W. BARBER, M.B.

J. G., male, aged 46.. The disease began in 1923 with itching of the legs. In three months' time an apparently general exfoliative dermatitis had developed ("homme rouge"). The patient was under the care of Dr. McKenna, and was in a nursing home for ten weeks. In about a year the skin was clear. The next attack was in 1926, and began with itching of the back, but was not so severe as the first. The third attack was in 1927, and since then the skin has never quite cleared. The present fungating tumour and the subcutaneous nodular swelling near it were first noticed about six weeks ago.

When he consulted me, on January 3, 1930, there was a general dusky staining of the whole skin, such as is seen after an exfoliative dermatitis. Over

the lower end of the right scapula was a large fungating tumour, and to the right and below this, was a dusky red, deep, nodular swelling, to which the overlying skin was adherent. The lymphatic glands in the right axilla were much enlarged. The diagnosis clearly seems to be mycosis fungoides in which a generalized premycosic erythrodermia has now been succeeded by the development of tumours.

Discussion.—The PRESIDENT said he thought Dr. Barber's diagnosis of mycosis fungoides must be accepted. He had had a case which imitated—or actually was—psoriasis, and which developed into mycosis fungoides; but he had never before seen exfoliative dermatitis as a premycosic eruption.

Dr. H. C. SEMON said that another somewhat unusual feature of the case—the diagnosis of which he did not question—was the absence, in the general erythrodermia, of triangular areas of normal skin, a feature which in his experience was very characteristic in the premycosic stage of the disease.

Dr. GRAHAM LITTLE asked whether it was not the usual experience that the "homme rouge" stage was rapidly fatal.

Dr. BARBER: My experience of mycosis fungoides is that the prognosis is always grave.

Eruption (? Antipyrinides): Case for Diagnosis.—H. W. BARBER, M.B.

Patient, female, aged 53, unmarried. She is said to have had a rash on the feet two years ago, following an operation. On the arms the present eruption appeared first in March, 1929, when she was under a doctor and taking medicine. It cleared up, but relapsed six months later, and then cleared up again, but relapsed three months ago. She states that she has been taking no medicine.

She was first seen by me January 7, 1930, when there were circular or irregular patches of a brownish or bluish colour, and somewhat scaly, situated on the arms, wrists, backs of the hands, legs, thighs, buttocks and internatal cleft, and one on the back of the neck. According to the patient they were then in a quiescent stage, but were liable to become raised, red, and irritating. Their appearance suggested to me the specific eruption that may be caused by antipyrin, being very similar to that of an eruption undoubtedly due to this cause in a patient I saw two years ago. On January 10 I saw the patient again when the patches were, as she had described, congested, raised, and bright red. She had taken two tablets of a patent aspirin compound on the previous evening. On January 14 the patches had subsided, and were exactly in the same condition as at her first visit.

I am unable to suggest any other diagnosis except a drug eruption due to antipyrin or some allied compound, but I cannot obtain a satisfactory history in corroboration.

Discussion.—Dr. GRAHAM LITTLE said that some years ago he had seen a similar case. The patient had been taking, in Germany, a patent medicine called "salipyrin" a salt of antipyrin. The medallion-like lesion was practically restricted to takers of antipyrin compounds; at least, he (Dr. Graham Little) had not seen it in other patients.

The PRESIDENT remarked that the lesions were less inflammatory in appearance and more "fixed" than in a case of the kind he had seen; and he thought that similar fixed medallion-like plaques also occurred in phenolphthalein erythrosis.

Dr. H. C. SEMON said that he had seen one example of an eruption presumed to be due to phenolphthalein. When first examined it had strongly resembled pityriasis rosea, and the usual prognosis had been given. There were typically scaling medallions of fawn colour, etc., and the eruption was limited to the trunk. Gradually it spread to the neck and assumed a rather urticated character. Irritation increased and some of the lesions coalesced and ran into each other, forming circinate figures which reminded one of a tertiary syphilide. The patient sought a second opinion, and Dr. Whitfield, whom she consulted, said he thought the rash might be due to phenolphthalein, which she had been taking for years in a proprietary mineral oil laxative. The symptoms had cleared up on its omission, but there had been no opportunity of seeing whether they recurred on resumption.

Erythrodermic Pemphigoid Lichen Planus.—LOUIS FORMAN, M.D.

J. W. T., male, aged 43, a painter. Noticed rash on trunk August, 1929. Seen at Guy's Hospital in the beginning of November, 1929, when the condition was diagnosed by Dr. Barber and Dr. Dowling as acute lichen planus.

An injection of novarsenobillon—0·3 grm.—was given. A week later a diffuse erythematous indurated eruption appeared all over the trunk and limbs, formed by confluent papules, on which arose bullæ later. The condition has gradually improved but fresh lesions are to be seen on the back.

Discussion.—Dr. GRAHAM LITTLE said he regarded this condition as a dermatitis herpetiformis, with an accidental association with a past lichen planus. There was at present no evidence of lichen planus, at any rate clinically. He did not think the lack of improvement under arsenic constituted very positive evidence. It was not enough to invalidate the diagnosis of dermatitis herpetiformis.

Dr. WIGLEY said he would suggest tentatively that this was an unusual form of novarsenobillon dermatitis. The eruption had come out soon after the injection was given. He was aware that it was unusual for an eruption to follow one small dose of novarsenobillon, but it did occur occasionally. He had recently seen a case of a bullous eruption, more or less confined to the arms, legs, hands and feet, which had definitely followed several doses of novarsenobillon.

Dr. H. W. BARBER said that when he first saw this case there was no question about the diagnosis of lichen planus.

He joined issue with Dr. Graham Little, especially concerning the lesions on the back, where some bullæ were present. The non-bullous lesions here, however, were not like the non-bullous lesions of dermatitis herpetiformis; they were definitely grouped, flat, papules, and not the urticated papules of dermatitis herpetiformis. He agreed with Dr. Wigley that the injection of novarsenobillon probably had something to do with the transformation of the eruption, but it must be remembered that it was a characteristic of lichen planus sometimes to come out acutely after injections of arsenobenzol; an eruption of moderate extent might thus become generalized in forty-eight hours.

The PRESIDENT said that if he had not the assurance of Dr. Barber and Dr. Dowling as to the original diagnosis of lichen planus, he would agree with Dr. Graham Little that the eruption closely resembled dermatitis herpetiformis. He had seen a case of extensive papular lichen planus following the use of novarsenobillon, but he had never seen any bullous eruption following that drug. He could not in this case make out definite lichen planus papules, and he would have thought it unlikely for a bullous lichen planus to have occurred without some of the typical lichen planus remaining.

Section of Balneology and Climatology.

[*February* 21, 1930.]

DISCUSSION ON BATH REACTIONS IN SPA TREATMENT.

Dr. J. Barnes Burt: In its broadest sense this term refers to all the body changes which occur (1) immediately after a bath, and (2) after a series of natural baths. The subject is a large one, and I propose to deal with two phenomena only: (1) the thermal effects which immediately follow a natural bath; (2) the reaction pains which occur in various rheumatic and gouty conditions following a series of baths.

I. Thermal Effects.—In the literature of hydrology there are many references to reduction of temperature of febrile patients by means of baths, but very few to the increase of temperature by the use of baths. Kellogg and Baruch briefly refer to the rise of temperature following hyperthermal baths in Japan, and Bazett [1] very shortly discusses the temperature in neutral baths. Currie [2] in experiments on himself, showed that baths at an indifferent temperature produced no rise of temperature.

The following investigations, made chiefly on patients in Devonshire Hospital, Buxton, do not agree with these findings. In these experiments, patients suffering from various types of rheumatism have been placed in weak natural radio-active water (0·4) for ten minutes, the bath at the beginning having a temperature of 96° F. and cooling down to 94° F. after ten minutes. The temperature was registered in the mouth. In a certain number of cases the rectal temperature was taken, but, as Bazett pointed out, the rectal temperature has certain disadvantages, and the mouth temperature for practical purposes is sufficiently accurate. In a series of 209 consecutive baths the average rise of temperature just before the patient got out of the bath, was 0·8° F., at the end of twenty minutes, 0·3° F., and at the end of an hour, 0·25° F. In analysing these figures one or two interesting points were noticed. One case of subacute rheumatic fever, two cases of gout, and two cases of early infective arthritis showed a rise of above two degrees. A case of gout in a man aged 64 showed an average rise of 1·3° F. in the first seven baths. The patient then had a thermal crisis and the last five baths showed an average rise of 0·3° F. only.

Some of the patients were given tap water baths at the same temperature on days alternating with the natural baths, and again a slight rise of temperature was found, the rise being somewhat similar to that which occurs after natural baths, but with this difference; at the end of an hour the rise after natural baths was greater than that after tap-water baths.

Again, some of the patients were given baths at a higher temperature, namely, 100° F., cooling to 98° F. at the end of ten minutes, and here I was surprised to find that the rise of temperature immediately after a bath was about the same as after neutral baths. All these patients were suffering from some form of rheumatism, and it was important to compare their reactions to baths with those of a perfectly healthy individual. I was fortunate enough to obtain the help of a healthy medical student who carried out a series of careful temperature experiments on herself in both natural and tap-water baths. The rise of temperature corresponds closely to that of rheumatic patients.

Table I shows the thermal effects of neutral and warm baths, both natural and tap water, on rheumatic patients.

TABLE I.—RHEUMATIC PATIENTS.

Duration of bath 10 minutes	Average rise of temperature End of minutes 10	20	60
Natural immersion 96-94° F.	0·8	0·3	0·25
Tap water 96-94° F.	0·5	—	0·04
Natural immersion 100-98° F.	0·8	—	0·33
Tap water 100-98° F.	0·75	—	0·26

Table II shows the thermal effects of both natural and tap-water baths on a healthy person, and also the thermal effects of cold baths, both natural and tap water, on a healthy person.

TABLE II.—NON-RHEUMATIC HEALTHY SUBJECT.

Duration of bath 10 minutes	Average rise of temperature End of minutes 10	30
Natural immersion 96° F.	1·4	1·2
Tap water 96° F.	0·39	none
Natural immersion 80·5° F.	1·45	0·6
Tap water 80·5° F.	0·65	none

The obvious explanation of the rise of temperature after a neutral bath is that the evaporation of sweat is very much reduced, and as the temperature of the skin is about 95° F., practically no heat is extracted—hence the rise in temperature. This cannot be the entire explanation however, because with baths a little above skin temperature there is no greater rise of temperature, and after baths of a temperature as low as 80° F. there is still a rise of temperature. This suggests that cold baths of short duration produce an increased metabolic action, whereas baths pleasantly warm lead to the same result as neutral baths.

The additional heat found after taking a radio-active bath is possibly due to increased metabolic changes, the result of inhaling radon emanation, and the absorption through the skin of beta and gamma rays. This suggestion is supported by the length of time the temperature remains raised.

I should like to point out one or two difficulties. The ordinary person entering a bath of 96° F. unconsciously starts rubbing himself as he enters the water, whereas in a bath of 100° F. this does not occur. Another point is that the natural baths were given in a large room divided into numerous cubicles, open at the top and in front, whereas the tap-water baths were given in a small room without much ventilation. The tap-water baths could not be given in the deep immersion baths because the inhalation of radio-active gases from the other baths would have vitiated the results.

The figures I have given are perhaps too few to dogmatize upon, but they are interesting and suggest a fruitful line of research. One would like to see comparative figures showing the thermal effects of deep immersion in Droitwich brine baths, Harrogate sulphur waters, and Bath radio-active waters.

II. Reaction pains, sometimes known as bath fever or thermal crisis. It is one of the most interesting and important phenomena connected with spa treatment. Practically all types of rheumatism may develop reaction pains, but the phenomenon is shown *par excellence* in cases of true gout. In a typical case, generally after the third or fourth bath, there is a lighting up of joint pains, often associated with lassitude and mental depression, and, in a few cases, slight fever; in fact it resembles a slight attack of gout, the only difference being that the pain rapidly disappears without any treatment if no other bath is given during the period. When the reaction pains are over, it is advisable to continue the course of baths. Sometimes the bath fever does not occur till the end of the third or fourth week, and it is then known as a delayed crisis. Equally good results may be

obtained in such cases. Cotar [3], of Vichy, describes a somewhat similar phenomenon in cases of malaria undergoing spa treatment. Since working on this subject I have been surprised to find how often the physician misses the occurrence of reaction pains. In hospital there is a well-established tradition that "you have to get worse before you get better," and the patient, unless questioned very closely, will not volunteer any information about increase of pain, which he considers part of the routine. Two or three of my cases were only discovered by taking the bath temperatures. In other cases an ordinary temperature chart is the only evidence of bath reaction. Even amongst highly intelligent private patients it is not always easy to get information about reaction pains. These patients generally live in hotels and boarding-houses in which, as in hospital, the tradition of getting worse before you get better holds sway, with the result that the patient does not think it worth while to inform the physician. The increased pain is taken as a matter of course. I feel certain that the spa physicians miss many cases of reaction pains.

The various types of rheumatism vary considerably with regard to reaction pains. As mentioned before, true cases of gout show the condition in its most typical form, and in my own experience it is the exception for a case of gout not to show reaction pains. Even with the use of artificial radio-active waters, true gout shows definite reaction pains [4].

Patients who, though not actually suffering from gout, but have a definite family history of gout, are also particularly prone to marked reaction pains. Thus, in a case of gonorrhœal arthritis, in which there is a family history of gout, typical reaction pains are more likely to develop than in a case without gouty history. Reaction pains are regarded as a good sign; once these are over, the patient generally progresses steadily, and it is safe to promise a real improvement.

With regard to other forms of arthritis, the development of reaction pains is seldom so definite. They vary from stiffness all over to a fairly severe increase of pain in one joint, but in view of the chronicity of most of the conditions, subacute attacks like those which occur in gout are hardly to be expected. In climacteric arthritis reaction pains are quite common, not so much in the knees perhaps, as in other parts, the favourite site being the shoulder muscles and the trapezius, fibrositis of which is so often associated with the condition. In infective arthritis (gonorrhœal and focal) a few cases develop reaction pains, but the condition is not so marked as in gout. Anton Fisher says that out of sixty-nine cases of primary chronic polyarthritis, only five had a bath reaction, and of twenty-two cases of spondylitis ankylopoetica, bath reaction pain was only present in one. His statements are based, not on clinical observations, but on S.R. measurements. He says that bath reaction pains, of the existence of which there is no doubt, depend on the intensity of the irritation produced by the treatment, and quotes various authors in support of his statement. In fibrositis, particularly in people of gouty stock, reaction pains are met with. These may take the form of stiffness all over the body, or of an attack of pain in an entirely different region from that originally affected. In ordinary osteoarthritis, such as osteoarthritis of the hip, one has not noticed this reaction. Reaction pains are well recognized by spa physicians and various explanations have been suggested.

(*a*) Sir Alfred Garrod, who never practised in a spa, suggested that these reaction pains were due to the heat of the waters, and acted in the same way as a vapour bath in subacute rheumatic fever. He spoke from analogy and not from actual experience. In England, at any rate, many of our private patients have a hot bath every day in the year. They have no reaction pains in baths of ordinary water. In Buxton it is the natural water at a temperature of 82° and not the baths at a higher temperature, which most quickly brings reaction pains.

(*b*) Some writers have suggested that the packs, douches and massage associated with baths lead to the absorption of inflammatory products, and hence the reaction.

In Buxton at any rate, mere bathing in the natural baths brings on reaction pains more surely than any other bath.

(*c*) Anton Fisher explains the phenomenon as a type of non-specific therapy, in other words, protein shock, and this is the view held by most of the writers on the Continent. He gives as an example casein injections which produces an acceleration of S.R. followed by a retardation. This is an attractive theory, but still leaves entirely unexplained how baths of simple thermal water, like those at Buxton, can produce protein shock. Fisher does not really explain reaction pains, he merely classifies them.

With the introduction of the suspension stability test we now have a means of recording and measuring reaction pains or bath crisis. Anton Fisher (Aachen) was the first to use this method, and since the publication of his paper last August we are beginning to use this test for reaction pains at the Devonshire Hospital.

Suspension stability records ought to be taken each week, and on the occurrence of a drop, the patient should be closely questioned and examined. If this were done, many cases of reaction pains would be recorded which otherwise escape the notice of the physician.

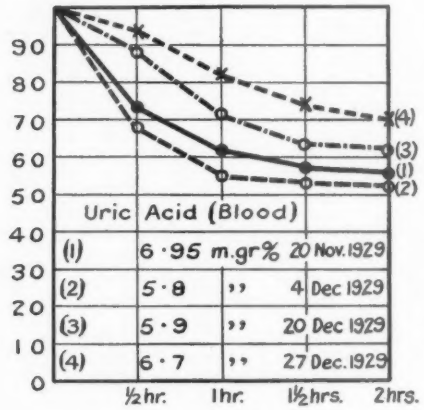


CHART I.

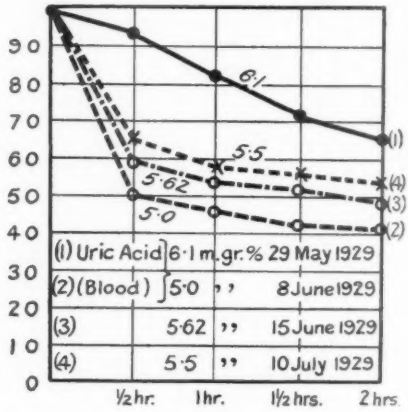


CHART II.

These two charts of cases of tophaceous gout illustrate the value of this test in studying reaction pains.

In Chart I the hourly suspension stability shows a drop of 6% coinciding with the increase of pain. Sixteen days afterwards there is a marked rise, and in twenty-three days the hourly suspension stability is 20% better, not only is the height of the suspension stability improved, but the character of the suspension stability curve is better, thus in the first half-hour it is almost normal.

It might be asked how these charts differ from those of a patient who has no reaction pains. If a patient had suffered from an ordinary relapse, due, shall we say, to injudicious treatment, the suspension stability remains at a low level for at least two weeks, and there would be no marked improvement at the end of a month similar to that shown in Chart I.

Chart II illustrates the difference between a relapse and reaction pain.

From the above illustrations it must be admitted that the suspension stability test offers a useful means of studying reaction pains, but the physician and the pathologist must work together, otherwise mistakes will arise. For instance, a cold

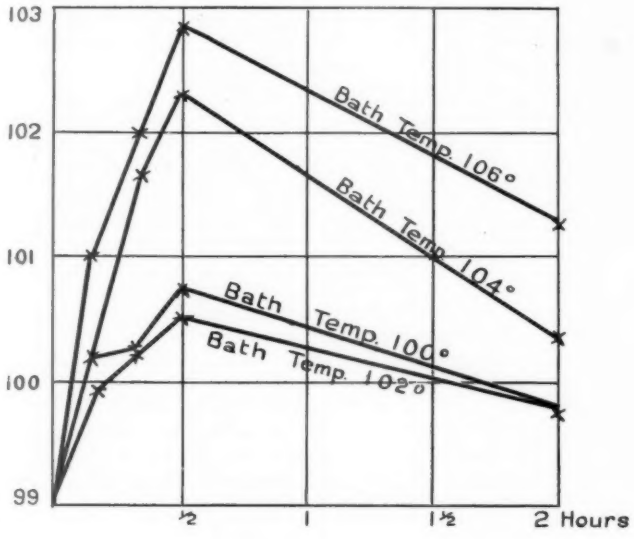
or a sore throat will produce a temporary drop in the suspension stability, and this might be interpreted by the pathologist as a reaction pain.

No mention has been made of subacute rheumatic fever, because in this condition a slight increase of pain, varying from day to day, is so common that it is extremely difficult to decide whether the patient has a reaction pain or not. However, a series of suspension stability tests will help the physician in deciding this problem, and in the course of the next few months, at the Devonshire Hospital, we hope to study the question with the help of this test.

REFERENCES.

[1] BAZETT, H. C., *Amer. Journ. Phys.*, lxx, 2. [2] *Medical Reports on Effects of Water Cold and Warm in Fever*, 1797. [3] COTAR, C., "The Mineral Waters of Vichy," 152. [4] PIERY and MILHAUD, "Les Eaux Minérales Radio-Actives," 147. [5] ANTON FISHER, "Blood Findings in Rheumatic Diseases," *Report of Research Institute for Rheumatism*, Aachen.

Dr. F. G. Thomson: The artificial pyrexia produced by immersion in warm baths must be an important factor in producing such effects as result from a course of treatment. Dr. R. G. Gordon and myself have been recently making a series of observations on the effects of baths at a higher temperature than those referred to by



Dr. Burt. The subjects of our experiments have been young, healthy adults, and in order to ensure accurate records we have used the Cambridge Instrument Company's apparatus for recording continuous temperatures in the rectum. Though observations made hitherto have not been sufficiently numerous to warrant our making any sweeping deduction, the accompanying chart showing the effects of immersion for thirty minutes at temperatures ranging from 100° to 106°, indicates that a very considerable degree of artificial pyrexia, lasting for at least two hours after the bath, is produced by this form of treatment.

The loss of weight, representing the loss of fluid by skin and lungs, resulting from an immersion bath at a relatively high temperature, is rather striking. By weighing the subject immediately before immersion and again two hours afterwards, it was found that the average loss was as follows:—

Bath at	100°	... ...	0·5 oz.
,,	102°	... ...	13·7 ,,
,,	104°	... ...	16·9 ,,
,,	106°	... ...	19·0 ,,

The increased metabolism associated with this artificial pyrexia is probably an important factor in producing "reaction pains," and suggests the importance of ensuring efficient elimination of waste products by the skin and kidneys during a course of treatment by immersion baths.

Dr. Geoffrey Holmes said he had made a number of estimations of the basal metabolic rate of healthy subjects after plain water and after peat whole-body immersion baths, at temperatures ranging from 103° F. to 110° F. In all cases the basal metabolic rate was practically the same half an hour after the bath as before it.

He had found that the physiological disturbances were more marked in the Harrogate saline sulphur baths than in plain water baths at the same temperature. In hot immersion baths he had found that immediate increase in pulse-rate and fall in diastolic blood-pressure were constant features. [Dr. Holmes then quoted examples of bath reaction caused by spa waters quite independently of the effects of manipulation.]

Dr. M. B. Ray said that the opinions of the mediæval physicians on the question of heat production and heat loss were of great interest in this connection. He quoted from the work of Avicenna (A.D. 980 to 1037) as set forth in the "Canon of Medicine" (Gruner's translation) to show that recent researches on the effects of hot and cold baths and the reactions following them, only confirmed views that had been currently held by medical writers for the last nine hundred years.

Dr. H. W. Hales: In connection with foam bath therapy for rheumatic conditions, it has been noted that the two essential reactions produced by this means are pyrexia and sweating. These are the results of the intake of heat over a small area, and the prevention of loss of heat by the insulating action of foam over a large area. It has been suggested that foam, being a saponine, may reduce surface tension of the skin and dissolve skin lipoids, and that colloids introduced into the foam in a high state of dispersion may be absorbed through the skin. The recent paper by Monod[1] is recalled in this connection. A close relationship is traced between intestinal toxæmia, thyroid deficiency and inactive skin. I have had cases in which this remedy has been successful when every other known method has failed.

A fall in systolic and diastolic blood-pressures is noted in all normal and high cases. Some patients with low blood-pressure have actually recorded a rise. A persistent fall in blood-pressure indicates the necessity of reducing the frequency of the treatments. The importance of pH values of sweat as a guide to progress under this treatment has also been noted. The presence of lactic acid in sweat is due either to deficient oxidation in the periphery or to failure of re-synthesis to glycogen owing to intestinal toxæmia, and deficiency in vitamin B. Sulphur foam may possibly act by improving the glutathione content of blood.

[1] *Lancet*, 1930, i, 227.

Dr. W. P. Kennedy: The frequent reactions, and fuller content of uric acid in the blood, at the termination of bath treatment, can be accounted for when we consider that: (1) The daily excretion of uric acid is a slow process (say 0·75 grm.), and another 0·75 grm. may be accounted for by destruction in the liver. (2) Uric acid has a great propensity to lie deposited in various connective tissues, and outside the blood-stream. Balneological treatment may dislodge great stores and account for marked uricacidæmia. From time to time during treatment there will be a tendency to fresh deposits, which will account for the frequent reactions, and at the termination of the treatment will explain excess of uric acid over that which was present before treatment. The blood tolerates large accumulations, and possibly the patient experiences more buoyant feelings from that very fact.

As to internal temperature, I would expect a primary rise in rectal temperature from a subthermal bath.

Among baths differing in temperature, I would like to emphasize the high therapeutic value of the indifferent baths (about 95° F.). Schuller records general contraction of the vessels of the pia mater and a sinking of the tissues of the brain in trephined animals, in the indifferent bath. The calming effect of the prolonged tepid bath in great mental excitement is well recognized. A marked soothing of the peripheral nerve-endings is likewise acknowledged.

In the evolutionary development of animals, from the poikilothermal to the homoiothermal state, we can trace the advancing efficiency of the complicated nervous mechanism controlled by the thermotaxic centre, and not only can we hope to influence the control of the bodily heat and metabolism, but we can also give an explanation of the faith that is in us in our regulation of such mechanism.

Section for the Study of Disease in Children.

[*February* 28, 1930.]

Report on a Case of Purpura Hæmorrhagica following Diphtheria.—J. D. ROLLESTON, M.D., and D. G. MACPHERSON, M.B.

A boy, aged 4 years, was admitted to hospital with moderately severe faucial diphtheria on December 16, 1929—the sixth day of the disease. A single injection of 24,000 units of antitoxin was given on admission, and the throat became clean on December 23. Suppurative cervical adenitis developed on December 24. On January 3, 1930 (twenty-second day of disease) an urticarial serum rash appeared on the limbs, with swelling of the scrotum, due to fluid in the tunica vaginalis. On January 8 (twenty-seventh day of disease) double otorrhœa developed, bleeding took place from the mouth and pharynx and the stools were black. The

KIDNEY.

STOMACH.

Case of purpura hæmorrhagica following diphtheria.

[We are indebted to Dr. J. E. McCartney for these photographs.—J. D. R., D. G. MacP.].

following day the child showed generalized petechial hæmorrhages and purpuric patches on the abdomen and loins, as well as hæmorrhages from the mouth, pharynx, and rectum. The heart, which had hitherto been normal, developed a cantering rhythm, and death took place on January 10 (twenty-ninth day of disease). There was no albuminuria, but no urine was passed for twenty-four hours before death. The temperature during the child's stay in hospital had ranged between 99° and 102°.

Necropsy.—Pharynx and œsophagus showed numerous small hæmorrhages. Trachea normal. Stomach: numerous hæmorrhages in submucous coat and on external surface. Intestine: numerous hæmorrhages throughout small and large intestine. Liver: a few small hæmorrhages on surface. Pancreas: normal.

Kidneys: numerous large hæmorrhages throughout cortex and medulla. Bladder: contained a little blood-stained urine and showed some small submucous hæmorrhages. Heart: numerous small hæmorrhages throughout the myocardium. Brain: anæmic. No hæmorrhages seen.

Microscopical sections of the stomach, intestine, liver and kidneys (kindly prepared by Dr. J. E. McCartney) showed toxic changes, especially the kidneys, in which, in addition to the necrotic changes, there was a marked infiltration, with plasma cells and large mononuclear cells.

This case is reported for two reasons: (1) because of the extraordinary rarity of purpura hæmorrhagica in diphtheria, apart from the acute stage; (2) the possible relationship of the appearance of the eruption to antitoxin. It is an extremely rare event to follow diphtheria, i.e., during convalescence. Only four cases of the kind have been recorded; one by Dr. Buckley,[1] and three by Dr. Goodall,[2] and they all recovered. This is the first case following diphtheria that I have seen. In 1915, Dr. E. B. Gunson[3] showed to the Section a case of what was practically purpura simplex, though it was a very severe form to which Henoch had given the name "purpura fulminans." In that case the purpura appeared some time after the serum rash. One is reluctant to attribute purpura, especially fatal purpura, to antitoxin, and particularly so in the present case, because though twenty-five other patients had the same brand of antitoxin, none of the other twenty-four showed any purpura. Purpura has been recorded in one of Dr. Goodall's cases in which no antitoxin had been given, as it was in the pre-antitoxin era.

Discussion.—Dr. E. W. GOODALL said that it was nearly thirty years since he had published the case to which Dr. Rolleston had referred. The patient in that case had urticaria, followed by erythema circinatum, and the question discussed was whether the purpura might or might not be due to the serum. As he had seen purpura following diphtheria in pre-antitoxin days, he did not feel justified in saying it was due to the antitoxin in that case; and he did not see any reason to alter his opinion now. He had not seen a similar case following diphtheria. He had, however, twice seen such a sequel follow scarlet fever, after which it was not so rare as after diphtheria. Both the cases to which he referred had been fatal; the condition was purpura fulminans, and death took place within about forty hours.

He asked whether concentrated serum had been used in this case, such serum as it did not cause serum sickness so often, or of such a severe character as did whole serum.

Dr. ROLLESTON (in reply) said that a concentrated serum had been used. For some years concentrated serum only had been used at his hospital, and serum phenomena had been much rarer since it had been employed; it was now unusual to meet with any sequels, except urticaria. The case also showed what had only once been recorded in this country,[4] though several French writers had reported it, namely, effusion into the tunica vaginalis, possibly because of involvement of the testicle. Purpura fulminans was much more commonly found after scarlet fever than after diphtheria. In thirty years he had seen about six such cases following scarlet fever. Although, unfortunately, no blood examination had been made, meningococcal infection could probably be excluded in the present case.

Congenital Melanotic Sarcoma in an Infant.—F. PARKES WEBER, M.D., E. SCHWARZ, M.D., and R. HELLENSCHMIED, M.D.

Specimen: Liver showing nodules of melanotic sarcoma from a child, aged 10¼ months, whose mother died from melanotic sarcoma three months after delivery. The placenta was melanotic, according to Mr. Eardley Holland, who delivered the child by Cæsarean section (at term) at the London Hospital. The child, who at first had developed normally, was admitted to the German Hospital at the age of

1 C. W. Buckley, *Lancet*, 1901 (ii), 132.

2 E. W. Goodall, *Guy's Hosp. Rep.*, 1894, l, 97; *Trans. Clin. Soc. Lond.*, 1898, xxxi. Rep. Committee on Antitoxin of Diphtheria, Appendix I, 34; *Lancet*, 1901 (ii), 1492.

3 E. B. Gunson, *Proceedings*, 1915, viii (Sect. Dis. in Child.), 55.

4 Goodall, E. W., *Brit. Journ. Child. Dis.*, 1925, xxii, 39.

8 months, when the liver was felt to be enlarged, with tumour-like nodules projecting from the anterior surface. The case is apparently the first reported one of transmission of a malignant neoplasm from mother to child by intra-uterine inoculation. The specimen has been presented to the Museum of the Royal College of Surgeons of England.

Discussion.—Dr. STANNUS asked in what situation the primary growth in the mother was found.

Dr. GOODALL asked how long the mother was ill before the first sign of sarcoma appeared, and how long she had the disease before she died.

Dr. PARKES WEBER (in reply) said that the mother had been operated upon eighteen months before the delivery of the child, for melanotic sarcoma of the thigh. The placenta had been examined by Professor H. M. Turnbull, and was found to contain melanotic sarcoma. In regard to the question as to whether the growth in a given case was really melanotic sarcoma, or melanotic carcinoma, the difficulty could be avoided by using the term "malignant melanoma."

Gastromegaly in a Child aged 2 years.—REGINALD MILLER, M.D.

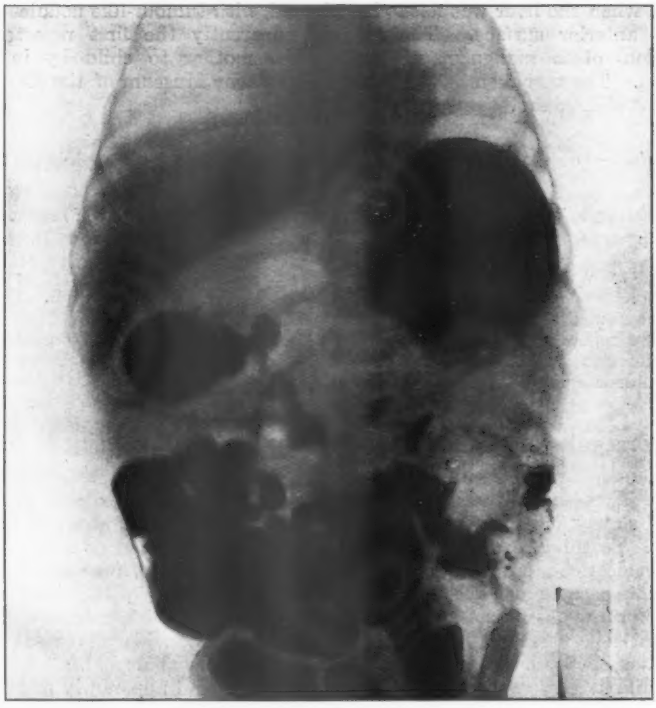
R. W., female, born February, 1928, at eight months, weighing 4½ lb. Never breast-fed. Projectile vomiting for first two months. After this ceased, she progressed slowly and took her feeds well. At the end of the first year she weighed 13 lb. 6 oz. Teething began at the age of 11 months, and she became cross and restless, but was not sick. She suffered from perpetual gastric flatulence, but not from hiccough. Constipation became severe during the second year, and she gained very little weight. For the last three months she has gone right off her food, and has lost weight. At 24 months she weighed 13 lb. 11½ oz. She has never sat up. The abdomen has always been large, but was not regarded as abnormal.

The child is very small and thin, but quite placid and apparently free from pain. Vomiting is only occasional.

The abdomen is very large, and the greatly distended stomach can be seen crossing the upper part. Following gastric lavage, which has removed a large amount of mucus, gastric peristalsis has been observed. X-ray examination shows the distended stomach, passing far to the right and hiding the duodenal cap. There is a large food residue at 7½ hours, this being chiefly in the cardiac end owing to the fact that the child was photographed lying on her back. The duodenal cap is not seen. (See fig. p. 34). The colon is voluminous, and a barium enema of two pints is easily delivered.

Suggested Diagnosis.—There is a congenital obstruction to the evacuation of the stomach. As the duodenal cap has not yet been seen, it is as yet impossible to exclude the pylorus as the site of the stricture. It is more likely, however, that the obstruction is at the duodeno-jejunal flexure, and if so, it is more probably due to arterio-mesenteric compression than to congenital stenosis or obstruction by band.

There are cases of gastromegaly in which there is arterio-mesenteric compression of the duodenum from birth. There are very severe cases which have caused death in the first few weeks, and in which post mortem there has been discovered this compression with an enormous duodenum behind it. The present case was probably of the same type but of less severe grade. It would be open to anybody to believe that it is an example of the colonic type of duodenal ileus, and that the compression of the mesenteric pedicle is due to the drag, not by visceroptosis or gastroptosis, but by the distended, dilated, and possibly floating colon. The result of a barium enema in a very small child is, however, always difficult to interpret.



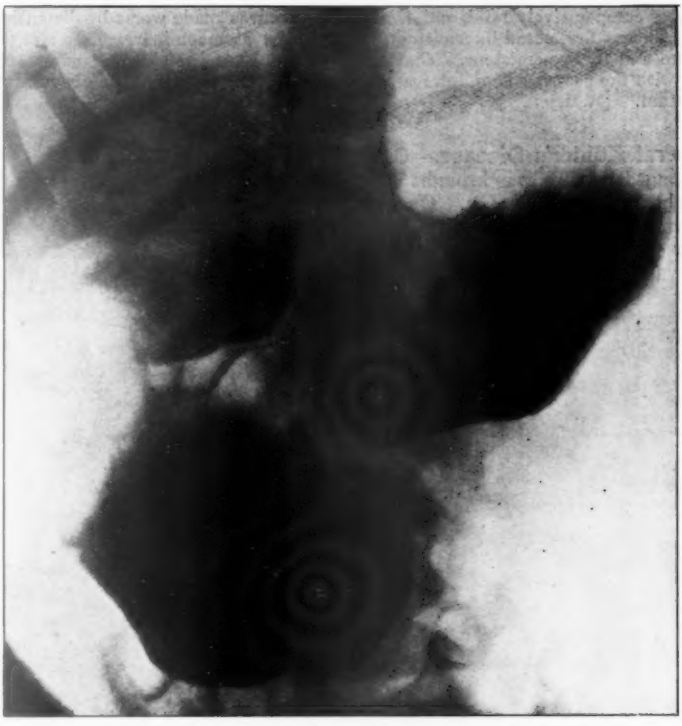
Skiagram showing greatly enlarged stomach with residue of an opaque meal, two and three-quarter hours after ingestion. The residue appears divided into two parts because the patient is lying on her back. (Dr. Miller's case.)

Discussion.—Dr. ALAN MONCRIEFF said that he had brought (for Dr. Poynton) a girl now aged 3, whose case was similar to the cases referred to by Dr. Miller, and in this instance there was a curious large duodenal pouch. As shown by X-rays, the meal went straight into this duodenal pouch. Mr. Twistington Higgins operated, and found the whole duodenum dilated to the middle of the third part, but there was no obstructing band and no evidence of any dragging by the mesentery. Mr. Higgins thought there was congenital atresia in the actual wall of the duodenum. Mr. Higgins anastomosed the jejunum into the sac and all vomiting ceased. Further X-ray examination showed that the sac still persisted, but the meal was now retained normally in the stomach.

Mr. ERIC CROOK said he would like to know whether bile was present in the vomit in Dr. Moncrieff's case. What would enable one to localize the site of the obstruction. When obstruction was below the pylorus, bile had been in the vomit. He believed it to be a way of distinguishing pyloric stenosis from duodenal obstruction. If there was no bile the site of obstruction was probably the pylorus.

Dr. E. A. COCKAYNE said he did not think Mr. Crook's criterion a safe one. He (the speaker) had described a case of atresia of the duodenum without even a fibrous cord joining the two parts, but with the bile-duct entering the distal portion. One case had been reported in which the bile-duct divided, sending one branch into the duodenum above and the other below the occlusion.

The PRESIDENT asked whether the dilatation of the colon was important in diagnosing the cause of dilatation of the stomach. He understood that in Dr. Moncrieff's case there was no obstruction in the stomach.



Gastromegaly. (Dr. F. J. Poynton's and Mr. Twistington Higgins' Case: see remarks by Dr. Alan Moncrieff.)

Dr. MILLER (in reply) said there was bile in the vomit in the case he showed. He was much interested in Dr. Moncrieff's case of megaduodenum. Three causes had been found for such cases. One type was congenital atresia. A second type was due to a band stretching across the duodenum. A third type was due to the superior mesenteric artery compressing the duodenum.

The point about bile in the vomit was not so simple as it seemed at first sight. If there existed serious obstruction in the duodenum, bile would be vomited in large quantities, but in the cases of which his present case was probably an example the obstruction to the duodenum was not very severe, and probably became complete only intermittently. A child of this type might vomit two or three pints, and the bile might be masked. In this case he thought bile would be recognized in some vomits, but not in all.

As to the colon, he was in the dark. In most cases of gastromegaly the colon was of normal size.

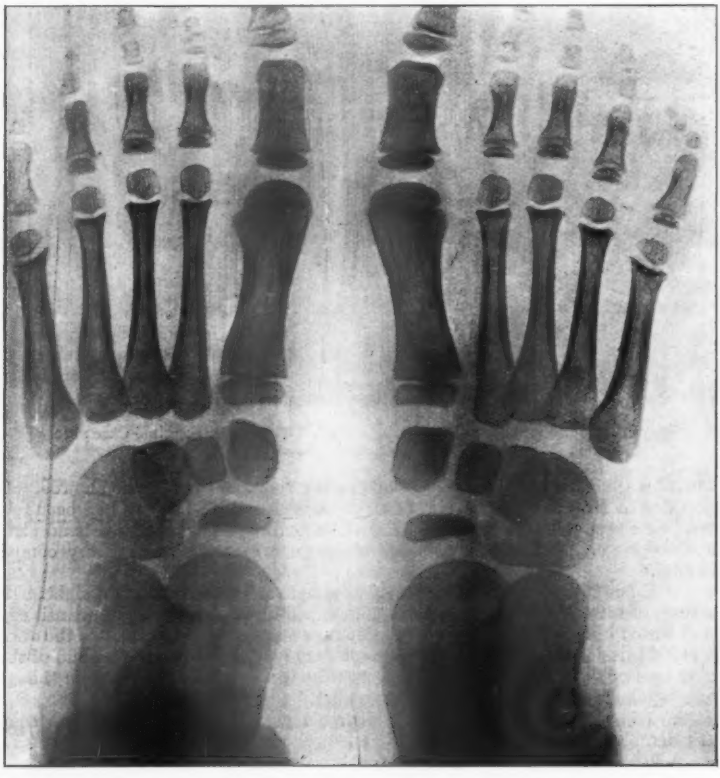
POSTSCRIPT.—The child died from vomiting and infective diarrhœa. At autopsy the stomach was found to be immensely enlarged, having a capacity of 10 or 12 oz. In spite of its increased size hypertrophy of the wall of the stomach could be appreciated with certainty. The œsophagus showed no hypertrophy. The pylorus was normal. The duodenum could not be recognized as either enlarged or hypertrophied. The colon was normal in size. Considerable gastritis and a very intense duodenitis were present. No obstructing bands were found. Evidence, therefore, of obstruction was clear, but the autopsy did not display the site or the nature of obstructive factor. It was certainly not in the stomach itself. Probably it was at the duodeno-jejunal flexure and due to arterio-mesenteric compression which is said often not to be demonstrable post mortem. The congenital obstructive factor

was probably comparatively mild, and the obstruction was made worse by distension of the stomach. This at first acted intermittently, but as the stomach increased in size it became more severe and more persistent. That there should have been such obvious duodenitis combined with the gastritis is of interest, as supporting the view that the obstruction was in the duodenum. [R.M.]

Bilateral Köhler's Disease.—CECIL P. G. WAKELEY, F.R.C.S.

P. D., aged 6 years, was brought up to hospital by her mother on account of pain in her right ankle due to a fall. On examination there were no physical signs. The movements of both ankles were full and free.

X-ray examination revealed bilateral tarsal scaphoiditis. The child was treated for genu varum at the age of 2 years, but there are no other evidences of rickets.



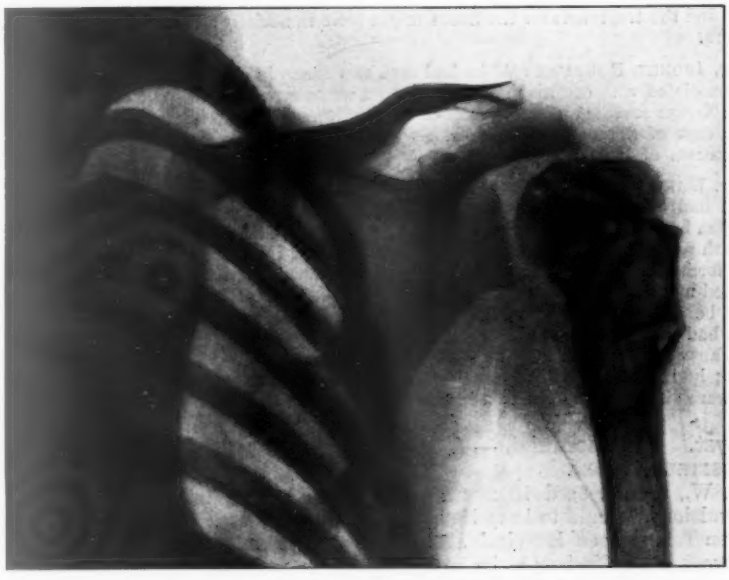
Bilateral Scaphoiditis. The skiagrams also show double epiphyses for the first metatarsal.

Fibrocystic Disease of the Upper End of the Humerus.—CECIL P. G. WAKELEY, F.R.C.S.

John B., aged 3 years and 8 months, brought to hospital February 11, 1930, on account of pain and loss of power in the right shoulder. The pain had come on quite suddenly while the child was playing on the floor.

On examination there was some slight thickening of the upper part of the right humerus, and some definite wasting of the right deltoid. Movements were limited.

On X-ray examination of the upper end of the humerus there was seen a clear area with well-marked borders, limited to the upper end of the humerus, and not traversing the epiphyseal line. There was some trabeculation across the cyst, and a line of fracture could be seen. A slight periosteal reaction was observed.



Fibrocystic disease of the upper end of the humerus.

The arm has been splinted in abduction, and the question is whether a surgical operation should be performed or the cyst in the humerus be left to consolidate. As the fracture has not completely traversed the cyst, I think an operation will be necessary.

Discussion.—The PRESIDENT said that Mr. Wakeley's first case was the kind that one wanted shown at these meetings because it was desirable to know what were the early changes in Köhler's disease as well as to be able to diagnose obvious cases of it. He had some hesitation in agreeing with the diagnosis, and he would "wait and see"; it would be interesting to examine further skiagrams of the condition. Five was the earliest age to see Köhler's disease, though a younger one was shown at the Section of Orthopædics by Miss Jebens. He had one at the moment which he was watching and treating as a tuberculous condition, as he was not sure of it. The ossific centre had disappeared, and then began to reappear.

He did not think that in Mr. Wakeley's second case a cure would result from the healing of the fracture. Operation would be needed, but it would be wise to wait and allow the fracture to unite, not only in order to see whether the reunion would heal the whole cyst, but also to give time for new bone to form between the cyst and the epiphyseal line. He had recently seen a case which supported that suggestion. The doctor thought it would be wiser not to have any operation done when first the bone gave way, as it would interfere with growth. Now, six months later, there was ¼ in. of bone between the epiphyseal line and the

cyst. In the meantime the bone had been broken again twice, and operation was now imperative. The fractures had caused some consolidation, but that had not cured the cyst.

Mr. ERIC CROOK asked whether Mr. Wakeley had carried out treatment by merely crushing the bone in, so converting it into a comminuted fracture, and stimulating the production of new bone. Would it not help the process if the fracture were made more extensive, to stimulate more bone formation?

Dr. J. KINGSTON BARTON asked whether treatment of the genu varum in the first case might not have been the cause of the bilateral Köhler's disease. The child had a very high instep and the treatment for the knees might have materially altered the lines of transmitted pressure.

Mr. LIONEL NORBURY said he had seen two cases, in adults, in which fibrocystic disease was associated with osteitis deformans. One was a case of fibrocystic disease of the tibia in which X-rays revealed a condition of osteitis deformans of the femur, skull, etc. The other was a case of fibrocystic disease of the tibia, in which the femur, too, showed the X-ray appearances of Paget's disease.

Mr. WAKELEY (in reply) said he would bring the first case up again in a year. The genu varum had been treated only at a cottage hospital, and he was now doubtful whether that condition had really been present.

With regard to the second case, he was interested in the President's remarks about new bone formation. He thought this conception was responsible for these cases not being operated upon early. He would not care to make a comminuted fracture, as he felt there might be some shortening of the bone afterwards; he would like to see a series of cases which had been so treated before deciding on such a form of treatment.

In answer to Mr Norbury, he thought fibrocystic disease in children differed from that in adults; he had shown adult cases [1] before the Clinical Section in which many bones of the body were diseased. In children it was more common to find only one bone involved.

Cyst. ? Intracranial. —J. MINDLINE, M.R.C.S., L.R.C.P. (for HUGH THURSFIELD, M.D.).

O. W., female, aged 10 months. Admitted to hospital December 9, 1929, with "convulsions." Said to have been always delicate and difficult to feed, and was in London Temperance Hospital at the age of 2 months on account of marasmus. One week after discharge she was dropped when being carried and the left thigh was fractured (August, 1929). There was no loss of consciousness and no bruising of the scalp at this time, and on readmission to the London Temperance Hospital no paralysis was noticed. She was discharged apparently quite well. In October, 1929, began to lose weight and to vomit. One week before admission to the Hospital for Sick Children, Great Ormond Street, began to have convulsions affecting face and all four limbs. On admission she was comatose. After two lumbar punctures the fits ceased; cerebrospinal fluid was under pressure, but otherwise normal.

On examination she was found to be much undersized (weight 10 lb.). There was paresis of right arm and hand with some rigidity, thumb being flexed into palm. All extremities were blue and cold, especially right hand, but there was no wasting and the reflexes were all present. Optic discs normal. Wassermann reaction, negative. Temperature rose to 101° F. three days after admission, but has been otherwise normal. January 22, 1930: An endeavour made to obtain blood from longitudinal sinus, needle being passed strictly in midline at posterior angle of the anterior fontanelle. About 10 c.c. of yellow, slightly blood-stained fluid were obtained; this had a high protein content (2 per cent.). Skiagram of skull showed no abnormality. Serum calcium 9·3 mgm. per 100 c.c (normal). Some improvement in condition of arm and hand since this fluid was removed. No further convulsions. The child is unable to sit up, and seems mentally subnormal.

[1] *Proc. Roy. Soc. Med.*, 1927, xxi, 267 (Clin. Sect. 19).

Dr. W. G. WYLLIE said there was very little choice of diagnosis in this case. It was one either of intracranial cyst or of porencephaly. Seeing that fluid had been obtained in the region of the anterior fontanelle, it probably came from a cranial cyst, as a porencephalic cyst was likely to be at a lower level near the island of Reil. The accident in August and the absence of symptoms until two months later fitted in with cases of dural hæmorrhages in adults in which it took two or three months, or even longer, for symptoms of epilepsy or delayed cerebration to appear. His diagnosis was, therefore, intracranial cyst.

As the cyst had already been tapped and considerable fluid evacuated, one would be tempted to leave it alone, but an exploratory operation would not be a serious procedure.

Paralysis of Right Upper Extremity following Accident.—ERIC I. LLOYD, F.R.C.S.

Boy, aged 2 years and 10 months, knocked down by motor-car, June, 1929. Details of accident lacking, but patient was unconscious for forty-five minutes. No vomiting or convulsions. Accident was immediately followed by paralysis of whole right upper extremity, and this has persisted.

First seen at Great Ormond Street Hospital, January, 1930. Rhomboids, spinati, teres major and serratus magnus are unaffected; all other muscles of the limb are paralysed, and do not respond to faradic current. Limb anæsthetic except for intercosto-humeral segment. X-ray examination negative. Sympathetic nervous system not involved.

The PRESIDENT said he had operated on five patients whose nerves had been injured at birth, as well as on others injured after birth. In the traumatic cases resulting from motor-car accidents the injury was usually very extensive, and of the "whole plexus" type. As a rule these cases were practically hopeless. In most of them the lesion was close to or inside the neural canal. In his last case the fifth nerve was found to be damaged; it was obviously fibrous outside the vertebral canal. The 6th, 7th and 8th were practically normal, i.e., up to the neural canal, but failed to give any response to stimulation. The nerves in these cases were pulled out by the roots from the cord, and in most cases the sympathetic was affected.

The present case seemed more hopeful, as there appeared to be a chance of finding the lesion amenable to surgery in at least some of the nerves. Boyer had reported a case, years ago, in a woman who, after her death a long time subsequent to the injury, was found to have even the opposite side of the cord damaged, the nerves having been pulled out by the roots.

Coarctation of the Aorta.—W. G. WYLLIE, M.D.

E. S., a girl, aged 5 years and 6 months. Cardiac and vascular condition noted on routine examination. No subjective symptoms. The heart is enlarged to the left (confirmed by skiagram). Heart sounds loud and forcible. Systolic bruit audible over præcordia, louder towards base. No thrill. Marked suprasternal pulsation, but no evidence of collateral circulation.

Blood-pressure in arms = 170 mm.; pulse big. No pulsation felt in femoral or dorsalis pedis arteries. There is possibly more than one deformity in this case. The basal systolic murmur suggests subaortic stenosis. The narrowing of the aorta presumably is beyond the origin of the subclavian, as the pulse in the arms is so easily felt.

Discussion.—Dr. PARKES WEBER said he agreed with Dr. Wyllie that this case was one of stenosis (coarctation) of the aortic isthmus. Possibly some pulsating subcutaneous arteries of the collateral circulation would become palpable later on. In cases of aortic isthmus stenosis, a slight abnormality of the aortic valves had occasionally been discovered by post-mortem examination, a fact that might help to explain the systolic murmur at the base of the heart in the present case.

Dr. BERNARD SCHLESINGER agreed with Dr. Wyllie's diagnosis; he thought that he could definitely feel thickening of the subscapular artery in this case, which was additional evidence of coarctation of the aorta. He did not think it was necessary to presume that there was also a lesion at the aortic valve, such as sub-aortic stenosis. In a similar case which he (the speaker) had shown recently at a meeting of this Section[1] the basal murmur

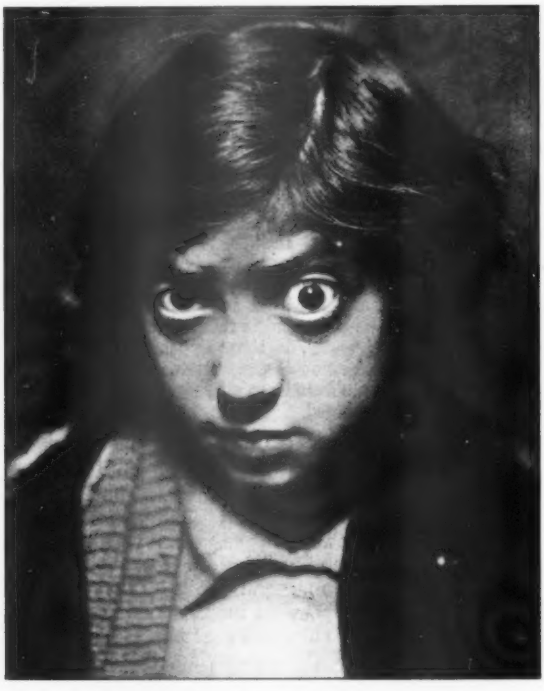
1 *Proceedings*, 1929, xxiii, 119 (Sect. Dis. in Child., 11).

and Corrigan's pulse were considered to be due to aortic regurgitation caused by the greatly increased blood-pressure in the aorta.

Dr. WYLLIE, in reply, said he considered that the pulse was well sustained and not of the Corrigan type.

Exophthalmic Goitre.—W. G. WYLLIE, M.D.

A. B., a girl aged 9 years. When 3 years old had ptosis of right eye; recovered in a short time. Broncho-pneumonia soon afterwards. After recovery from the pneumonia, left eyelid drooped for a few days. Pertussis at 4 years; scarlet fever at 5 years. At 6½ years, ptosis of the right eye followed by proptosis in a week's time and lasting several weeks. There is still slight exophthalmos of the right eye. Left exophthalmos occurred while the prominence of the right eye was diminishing and has persisted since.



Exophthalmic Goitre. (Dr. Wyllie's case.)

Present State.—Von Graefe's and Stellwag's signs present. Child is excitable, sweats easily and often shows hot patches of flushing on the body. Has frequently had urticaria since age of 13 months. Last attack was in January, 1930. She dribbles when excited or speaking rapidly.

Basal metabolic rate (Read's formula) = + 20. Blood-pressure 110/75; after rest 100/60. Pulse-rate 96, after rest 90. Blood-count: Red blood-cells 6,000,000, eosinophils increased. Protein skin reactions: positive to egg, shellfish, tomato, orange and feathers.

Family History.—One brother had bronchial asthma for twelve months when aged 16 years.

Discussion.—Dr. G. W. BRAY said that a fractional gastric analysis in this case had shown a hypochlorhydria, which was typical of most allergic conditions, therefore the absence of acid in the stomach might result in large quantities of undigested protein getting into the blood. Administration of acid medicines greatly benefited this condition.

Dr. BERNARD SCHLESINGER said he wondered why there was no tachycardia in this case.

Dr. H. S. STANNUS said that in allergic conditions and exophthalmic goitre the blood-calcium was usually low, and the low acidity diminished the amount of ionic calcium in the blood.

Dr. W. R. F. COLLIS asked whether Dr. Wyllie considered that the exophthalmic goitre was directly connected with the urticaria, or was a mere coincidence.

Dr. WYLLIE, in reply, said that the pulse-rate was perhaps normal now, but the chronicity of the condition and the fact that the child was improving might explain this. All the symptoms, the thyroid over-action, exophthalmos, urticaria, profuse salivation even to dribbling, he thought were closely associated in this case, and might be considered together as a neurosis of the vegetative nervous system.

Epituberculosis.—BERNARD SCHLESINGER, M.R.C.P.

R. A., a boy, aged 3 years, was brought for examination, complaining of slight cough and weakness for the last few months. Except for whooping-cough and pneumonia at the age of 1 year, from which he completely recovered, he had had no previous illnesses.

Skiagram of chest taken on October 30, 1929, showing opacity at the left upper lobe. (Dr. Schlesinger's case of Epituberculosis.)

Family History.—Mother had recurrent tuberculous pleurisy; one sister had tuberculous peritonitis, and three other children, the youngest aged 2 years, all gave positive reactions to Mantoux tests.

On Examination (July 8, 1929).—Impairment of percussion-note over middle zone of left lung in front and behind, with harsh breath-sounds over the corresponding area. Afebrile. Mantoux 1/10,000 positive. Sputum contained no T. B. Skiagram shows opacity at the left hilum. October 3, 1929.—Stationary weight. Physical signs more marked. Skiagram : opacity has now extended to left apex (see fig.). November 1, 1929.—Physical signs less marked. November 28, 1929.—Marked tubular breath-sounds at left apex and increased dullness. December 11, 1929. —Skiagram : opacity clearing up at left apex. Physical signs less marked. Sputum and fæces negative for T.B. Afebrile throughout except during a cold on one occasion, and when measles developed, December 31, 1929. January 24, 1930. —Dullness and tubular breath-sounds more marked, slight bronchitis remaining from measles.

February 14, 1930.—Dullness has extended downwards and crepitations are audible. Skiagram shows opacity at middle zone and apex is clearer. Heart not displaced. No T. B. in sputum. Weight stationary. Afebrile.

Discussion.—Dr. EDMUND CAUTLEY said that if the condition had been tuberculous, in a child of this age it would probably have become progressively worse and ended fatally. Experience showed that in children there could occur a slowly-extending broncho-pneumonic process which was practically afebrile and might last for months, or even for a year or two, and then clear up. In one case of this type he had seen such a condition involving the whole of one upper lobe and lasting about two years, with all the physical signs it was customary to attribute to tuberculosis.

Dr. PARKES WEBER said that the question in this case was whether the broncho-pneumonia-like shadowing in the radiogram had any causal relation to the tuberculous infection (presumably at the adjoining part of the enlarged hilus shadow). The term "epituberculosis" (signifying the development of something *on the basis of a tuberculous lesion*) might be used to suggest that the broncho-pneumonia-like shadowing represented a kind of focal reaction in the neighbourhood of a tuberculous focus similar to that resulting from the injection of tuberculin.[1]

Dr. W. R. F. COLLIS said that some so far unpublished work was of interest in this connection. A clinic had been formed for infants aged from one year upwards, who had been exposed to tuberculosis in their homes, in which some member of their family had active pulmonary tuberculosis. They were examined by X-rays at weekly intervals after exposure, and tuberculin-tested at the same time by the Mantoux method. In some cases the condition called epituberculosis became apparent in the skiagrams after about six weeks from the calculated date of infection. At the same time they began to give a positive Mantoux reaction. The Mantoux phenomenon was an allergic reaction, and he (the speaker) suggested that this was also the case in epituberculosis, which was probably an inflammatory hypersensitive reaction spreading out from the hilus of the lung, in which a focus of infection was located. These cases often ran an almost afebrile course, though the onset was usually associated with fever.

In the present case, the fact that the child was sensitive to 1/10,000 old tuberculin was significant. He did not agree that these were cases of tuberculous broncho-pneumonia; post-mortem evidence was not to that effect. The sputum never showed tubercle bacilli at the time, though it was possible that later they might develop into pulmonary tuberculosis.

In epituberculosis the prognosis was good while in caseous broncho-pneumonia it was bad.

Dr. W. G. WYLLIE said that something was to be put forward in favour of the term "epituberculosis." Its originators made a post-mortem examination in one case, in which the patient, a child, had died, not from lung disease but from sinus thrombosis, and in the lungs there were found some scattered tuberculous foci of the right upper lobe, which had been dull under X-ray examination. Around these foci there was a gelatinous pneumonia, not suggesting tuberculosis. That was why it was called "epituberculosis." The authors had brought forward the term tentatively for the purpose of grouping certain cases, and directing attention to them.

[1] I have compared certain transient recurrent attacks of supposed "pneumonia" (all in the same part of the lung) in adults, in regard to the theory of "epituberculosis." See *Proc. Roy. Soc. Med.* (Sect. Dis. in Child.), 1929, xxii, 27. [F. P. W.].

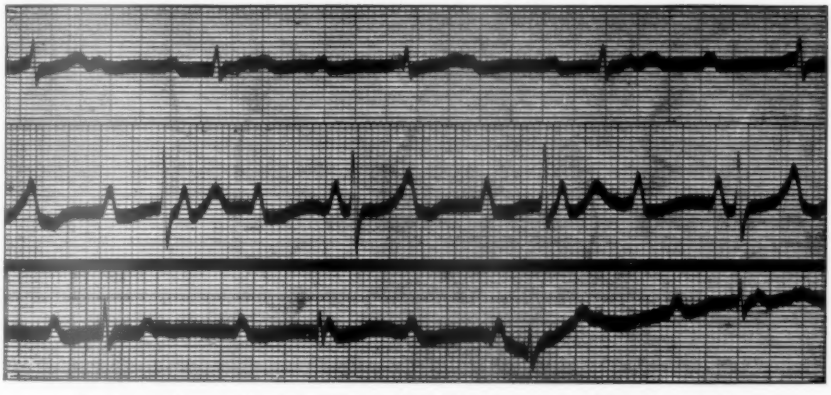
Dr. SCHLESINGER, in reply, said that this had appeared to be a case of unresolved pneumonia, but that would obviously have entailed a preceding pneumonia. This had not been the case, and since the condition bore all the credentials of so-called epituberculosis, this was the diagnosis made. In the one post-mortem examination that had been reported in the literature there was evidence of a non-specific pneumonia, and in the centre of it a caseous tuberculous gland.

He regarded this type of pneumonia in the same class as the lung changes often encountered in rheumatic pericarditis. They were pneumonic in nature, possibly brought about by a flood of toxic antigen acting on sensitized lung tissue and producing this remarkable inflammatory reaction. Experimentally, Bigelow[1] had produced a pneumonia of this allergic type. He did not think that his case had any very active tuberculous focus.

Complete Heart Block.—JOHN GIBBENS, M.R.C.P.

Patient, a girl, aged 10 years. *Past History.*—Normal labour; perfectly well at birth; no cyanosis or dyspnœa. At the age of 6 years attended Guy's Hospital Out-patient Department for a period of six months, complaining of dyspnœa and pain in the region of the heart. She would get a sudden stitch while walking; after she had stood still for a short time this passed off. Unfortunately no records are available of this illness. Has had measles and whooping-cough. No diphtheria or scarlet fever. Has always been well and played games like a normal child without any obvious dyspnœa.

In January, 1929, she had tonsillitis, lasting two weeks. Three weeks later she had a bad cold and a temperature of 101°. Slight pain in left iliac fossa; no chest pain or sore throat. Four days later was seen by a doctor who noticed a systolic murmur and sent her up to hospital.



Electrocardiogram showing complete dissociation between auricle and ventricle.

Condition on Admission.—Pulse 44. No enlargement of heart. Soft apical systolic murmur; not conducted. (?) Reduplicated second sound at apex. No clubbing. Blood-pressure: Systolic 100; diastolic indefinite. Entirely free from symptoms. Electrocardiogram: Complete heart block.

In view of the presence of complete block possibly following the febrile illness of the preceding week, she was kept on her back for one month, and an additional fortnight in bed. Pulse-rate 44; half an hour after subcutaneous injection of atropine, gr. $\frac{1}{100}$, rate was 69. Ephedrine hydrochloride, gr. $\frac{1}{2}$, had no obvious effect on the pulse-rate or electrocardiogram.

Discharged: well in health. Complete heart block.

[1] Bigelow, G. H., *Arch. Int. Med.*, 1922, xxix, 221.

April 2, 1929: Sudden attack of dizziness, cyanosis, and "wandering speech," lasting one minute. Ephedrine, gr. $\frac{3}{4}$ daily, ordered. May 15, 1929: Felt giddy while out in the road and fell over. No pain. Recovered in two or three minutes. June 6, 1929: Has had no more attacks of giddiness but has had some pain in the elbows and calves. Looks rather short and fat. Average weight for 9 years 10 months, 62 lb.; actual weight, $62\frac{3}{4}$ lb. Average height for 9 years 10 months, $51\frac{1}{2}$ in.; actual height, $49\frac{3}{4}$ in. August 18, 1929: Record of pulse-rate taken four-hourly, day and night, for three days. The rate only varied between 38 and 46. October 9, 1929: Pain in both shoulders and legs. No further attacks of giddiness. Living an entirely normal life. Never has dyspnœa on exertion.

January 9, 1930: Living normal life. Condition of heart unchanged. Height $50\frac{3}{4}$ in.

Two Specimens (Macroscopical and Microscopical) of Primary Carcinoma of Liver (Hepatoma).—R. C. LIGHTWOOD, M.D.

FIG. 1.—Primary carcinoma of liver (Dr. Lightwood's case). (A) Left lobe; normal in structure. (B) Right lobe, cut in half; primary carcinoma.

Female infant, aged 8 months, brought to the Princess Louise Hospital for Children, June 8, 1929, on account of a hard abdominal tumour discovered four months previously. Her mother had noticed swelling of the child's abdomen five months before. In spite of taking her feeds well the baby had been losing weight for six months.

Nothing of note in history; pregnancy had been uneventful; parents healthy; four other children, all quite well.

On examination: pale and wasted infant. Some small lymphatic nodes palpable in neck, axilla and groins. The abdomen was greatly distended, dilated veins being visible in a tense abdominal wall. The liver was much enlarged and could be bimanually palpated as a mass extending down into the right side of the abdomen with its lower border in the right inguinal region.

The baby went steadily downhill until death occurred six weeks later, August 16, 1929. At first there was a gain in weight, presumably due to the enlarging growth,

Date		Weight	Date		Weight
July 17, 1929	...	+ 11¼lb.	August 5, 1929	...	− 11¾ lb.
„ 20, „	...	+ 12 „	„ 9, „	...	− 11½ „
August 1, „	..	+ 12¼ „			

At autopsy the liver was found to be enormously enlarged. No secondary deposits were found, and, apart from the hepatic growth and some ascites, there was no abnormality. The kidneys, suprarenals and bones were examined without result. The left lobe of the liver appeared to be free from growth.

FIG. 2.—Section of right lobe of liver showing primary carcinoma. (Dr. Lightwood's case.)

Histological Report (by Dr. E. ff. CREED).—The section shows a primary carcinoma of the liver of liver-cell origin. The tumour consists of large rounded masses of cells, separated from neighbouring masses by coarse trabeculæ of dense fibrous tissue. In the trabeculæ there are many pseudo-bile canaliculi, and in one area in the section, apparently just beneath the liver capsule there is extensive deposition of fibrous tissue, embedded in which are very large numbers of canaliculi with some hæmorrhage. The cells present in the tumour are clearly all derived from the polygonal cells of the liver, but show a very varying degree of departure from the normal. In parts the resemblance to the normal structure is close, the nuclei are pale-staining and vesicular, and there is much granular cytoplasm, in some places with fat vacuoles. In other places the cells are smaller, with very little cytoplasm, and with densely staining nuclei showing many mitotic figures. The cells are mostly in large solid masses, but there is a tendency in places to arrangement in columns. There is some hæmorrhage and some blood-pigment is present. There is much necrosis with cyst formation.

This case is of interest, not only on account of its rarity, but also because the association of primary carcinoma and hepatic cirrhosis, limited to one lobe of the liver, raises an important pathological question.

Primary carcinoma of the liver usually occurs between the ages of 40 and 60; of these cases it is stated that over 80% have cirrhosis. Further it is usually held that cirrhosis is the underlying condition. Regeneration is believed to overstep the mark and to take on a neoplastic character. There are three possible explanations for the association of primary hepatic carcinoma and cirrhosis of the liver: (1) The cirrhosis may be the underlying condition and the growth result from it. (2) The growth may be primary and the cirrhosis a reaction to its presence in the liver. (3) Both the growth and the cirrhosis may result simultaneously as two types of pathological response to some unknown tissue irritant.

Limitation of both the growth and the cirrhosis to one lobe in the case under discussion makes one or other of the two latter explanations more acceptable.

Dr. PARKES WEBER said that in most adult cases in which primary carcinoma was present in livers which were cirrhotic, the cirrhosis was supposed to have preceded the carcinoma. The present case might be an analogous case in which only half the liver was cirrhotic. In extremely rare cases atrophy or cirrhosis had been found to be almost limited to the right or the left lobe.

Congenital Pulmonary and Mitral Stenosis.—K. TALLERMAN, M.C., M.D. (for A. G. MAITLAND-JONES, M.C., M.D.).

Patient, female, aged 11 years. *Family History.*—Negative. *Past History.*—Nothing significant except pneumonia at age of 10 years. Never noted to have been cyanosed as a baby.

History of Present Illness.—For three years mother has noted that child becomes cyanosed at times. For two years has suffered from pains from time to time in joints. Has been fidgety and on one occasion was unable to walk on account of pain. Has always been subject to slight coughs and to frequent sore throats. For the past year the appetite has been poor, and she has complained of abdominal pain and, for the past two months, of attacks of pain in the chest accompanied by blueness; these, however, have been becoming fewer.

On Examination (February 4, 1930).—Face shows a high colour over the cheeks but there is no obvious cyanosis; there is no clubbing of the fingers; temperature normal; pulse-rate 90. *Heart*: Apex beat in fifth space $\frac{3}{4}$ in. external to nipple line; cardiac dulness does not extend to right of sternum. In the pulmonary area a systolic thrill is felt and a systolic murmur is heard, conducted upwards and outwards. The pulmonary second sound is much accentuated. At the apex there is a presystolic and systolic murmur and the first sound is sharp and loud. *Lungs:* Some rhonchi; no other abnormalities. *Abdomen:* Liver edge palpable just below costal margin. *Tonsils* rather enlarged.

February 6, 1930.—Orthodiagram shows heart enlarged to left and right. Left auricle enlarged. Pulmonary artery much increased in size. Electrocardiogram shows right ventricular preponderance and a large P wave, otherwise nothing significant. Skiagram confirms the orthodiagram, showing the heart enlarged both to right and left.

February 20, 1930.—Child seems quite fit. Physical signs of cardiovascular system as before. No other physical signs.

Dr. SCHLESINGER said that possibly the condition in this case was not a congenital malformation, but was due to severe rheumatic carditis. Recently there had been attacks of pain in the chest and abdomen, which might have been caused by a smouldering pericarditis. An adherent pericardium was difficult to diagnose, but, for what it was worth, a diastolic shock could be felt at the apex in this child.

Section of Ophthalmology.

[*February* 14, 1930.]

Distichiasis.—J. H. DOGGART, F.R.C.S.

D. G., a girl, aged 7, was recently brought to Moorfields by her mother, who complained that the child's eyes had been red and watery for one year. There is no history of any other members of the family being similarly affected. On examination, all four lids are seen to have a row of accessory cilia arising from the posterior portion of the intermarginal surface. The accessory cilia are finer and fairer than those of the normal rows. They are more numerous on the lower than on the upper lids.

I have shown this case because of the rarity of distichiasis. Claes and Coppez [1] found records of only six cases in thirty years. Begle [2], writing in 1912, was able to collect altogether twenty-five examples from the literature.

The first histological examination of this condition was made by Kuhnt [3]. He demonstrated the absence of the Meibomian glands, which were replaced by an accessory row of cilia having sebaceous glands opening into their follicles. Brailey [4], Begle [2], and others, confirmed these observations. According to Whitnall [5] the Meibomian glands represent the ordinary sebaceous glands of a primitive secondary row of cilia which have disappeared in man. Blatt [6] and von Szily [7] apparently hold the same view, since they both regard distichiasis as a reversion to ancestral anatomy.

Several examples of a familial incidence have been noticed. Thus, Erdmann [8] recorded it in a girl whose mother and grandmother were both affected. Blatt [6] traced five examples in one family. Stanford [9] who reported a case in a boy, aged 6, discovered the same affection in the boy's mother and maternal uncle.

Distichiasis, although a congenital condition, does not usually cause symptoms in the first few years of life. Stephenson [10] reported it in a girl under 4 years, who had had symptoms for some considerable time, but most cases do not come for examination until much later than that. The delayed onset and the comparative mildness of the symptoms are attributable to the fine, downy nature of the accessory cilia, as contrasted with the coarse bristles commonly found in trichiasis. There are two other important clinical features of distichiasis: first, the almost invariable involvement of all four eyelids; secondly, the regular arrangement of the accessory cilia growing from the posterior portion of the intermarginal surface.

The treatment for this child will probably be electrolysis of the accessory cilia, one lid at a time, under general anæsthesia—a method that Brailey [4] adopted. Von Szily [7] performed an intermarginal section, and detached the posterior lash-bearing area by an incision parallel to the lid-margin. Begle [2] electrolysed some of the lashes, and dealt with the remainder by excision of a strip of posterior lid-margin, the gap being filled in by a graft from the lip. I would suggest that, since electrolysis offers an excellent chance of radical cure, it is unnecessary to mutilate the lids by a surgical operation.

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Discussion.—Dr. E. A. COCKAYNE said that he had asked the mother of this patient whether there was a family history of the condition, and she said a maternal aunt had it.

There seemed to have been very few cases in which the condition was hereditary; he had found only five or six such records. In these it had behaved as a dominant. A French author said that all the daughters during four generations had this condition, but none of the sons had it. This did not seem to fit the ordinary Mendelian theory; it was different from the case in which all the sons were affected in every generation, probably owing to a gene in the Y chromosome.

And there was a tendency for one type of the disease: three and four rows of hairs in some of the cases, and not just a straightforward transformation of the Meibomian glands into fine hairs, as in the present case. He had only seen one case before, in a girl, and in that there was no family history of the condition.

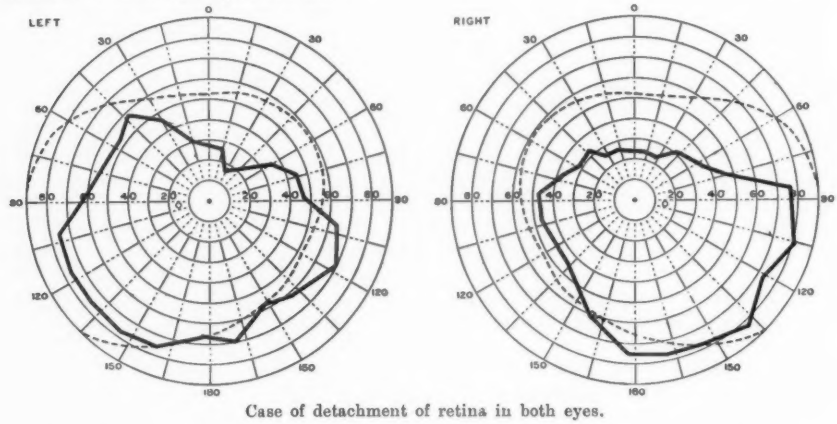
Mr. F. W. LAW said he remembered having to deal with a case of this kind about eighteen months ago. It was in a woman, who had had no symptoms until adult life. She was of dark complexion and there was a double row of lashes in one lid only. He (the speaker) had excised the accessory row of cilia. He had cut a V-shaped section, including the lashes, as far as he could, and then sutured. It was true there was a tendency to entropion of the normal row after operations such as this, but no entropion followed in this particular case. Six weeks afterwards there was no tendency to recurrence of the accessory row. The case showed the comparative ease with which accessory cilia could be dealt with.

Mr. DOGGART (in reply) said that the maternal aunt was not a case of distichiasis, but of trichiasis, resulting from old trachoma. She was attending the London Hospital periodically for epilation.

Detachment of Retina in Both Eyes.—A. F. MACCALLAN, C.B.E., F.R.C.S., and JEAN M. DOLLAR, M.B.

Patient, a boy aged 8, fell four years ago on some stone steps, wounding the left supra-orbital region. Previous to the accident, the sight appears to have been good, but afterwards he had to go on his hands and knees to find an object on the ground.

Visual Acuity.—Right eye, with sphere + 8 is $\frac{6}{36}$; left eye, with sphere + 8 and cylinder + 2 at 90 degrees, is $\frac{6}{36}$.



Case of detachment of retina in both eyes.

Ophthalmoscopic Appearances.—There is a detachment of the retina in each eye in the lowest part of the fundus. At the upper part of the detachment is a yellow band. Higher up there is a line of pigment parallel with the yellow band: this is probably the site of the former upper limit of the detachment.

The boy's general health is good; the tonsils are only slightly prominent. The Wassermann reaction is negative.

Double Detachment of Retina.—A. HUGH THOMPSON, M.D.

Patient, a boy aged 9, had 9 D. of myopia in each eye. In the right eye is an old detachment, which overhangs the central part all round. The disc can be seen, and has an ordinary crescent of about one-third disc diameter. In the left eye the detachment is not nearly so deep. It is deepest above; it is also seen on the inner side and below. The disc in the present case has an ordinary crescent, about the same as the other. There is a central opacity in the centre of each lens, not enough to interfere much with vision. There is some squint in the right eye. Vision: P. L. each eye.

The right eye was first affected in 1926. After that the boy went to a hospital as an out-patient, and had glasses prescribed there, minus 6 spherical with 3 cylinder for the left eye. The right eye, which was already blind from detached retina, had a plane glass. So there is no doubt that the right detachment dates from at any rate 1926. In August, 1928, Mr. Maddox signed a certificate stating that there was detachment of the right retina, and some atrophy of the left nerve, and that the patient could only count fingers, incorrectly, at one foot. He was, however, learning to read, up to the summer of 1929, at an ordinary school. In July or August last, however, he became blind. He then went to hospital again and saw Mr. Aynsley, who found that the left eye was squinting. (At present it is the right eye which is squinting.) Mr. Aynsley attributed the blindness in the left eye either to the squint or to possible nerve trouble. A skiagram was taken, but showed no abnormality of the pituitary body. The certificate is dated August 17, 1929, does not say anything about detachment on the left side. There is no doubt about the detachment now. I can find no hole.

Discussion.—The PRESIDENT said that detachments sometimes occurred in young people who were emmetropes. He remembered a case in which detachment had begun when the patient, a boy, was aged 9. When first seen, the detachment was in the lower part of the retina in both eyes, and there was much haze in the vitreous. The sight became worse and the detachment increased. Rest in bed and the administration of iodide of iron resulted in slight improvement. After some months he (the speaker) lost sight of the patient. Later he saw him selling newspapers in the street, but could not induce him to attend hospital to be examined. About six years afterwards—i.e., when 20 years of age—he was seen again after having been in France in the Army Service Corps. In spite of still having detachment in both eyes, he had about $\frac{6}{6}$ vision and was able to do good work. There were a certain number of these cases which belonged to a different group from those of ordinary detachment and were more like the case shown by Mr. MacCallan than that shown by Mr. Hugh Thompson.

Mr. ARTHUR GRIFFITHS said that two years ago he had exhibited a case[1] with lines of scar tissue, similar to those in Mr. MacCallan's case.

Retrobulbar Neuritis due to Concealed Nasal Sepsis.—ROSA FORD, M.B.

This is a further report of one of the cases I showed in October,[2] that of a woman with a pale disc and $\frac{6}{60}$ vision in her right eye. I bring it because a second operation has settled the diagnosis.

The evidence in October pointed to latent sinusitis as the cause of the retrobulbar neuritis, but doubt was expressed as to whether that diagnosis had been established, and also as to whether retrobulbar neuritis was ever due to inflammation of the sinuses, especially if these were ethmoidal and maxillary.

In the second operation the right side of the nose was approached extranasally, and it was thus easy to see the whole of the ethmoidal cells and the sphenoidals. It was evident that the sphenoidal and the anterior ethmoidal cells were healthy, but in the posterior ethmoidal region was a single cell in which the mucous membrane was thick and infiltrated. A portion of this cell-wall has been sent for

1 *Proceedings*, 1928, xxi, 1233 (Sect. Ophth. 49).
2 *Proceedings*, 1929, xxiii, 56 (Sect. Ophth. 14-16).

pathological examination, but as the bone has to be decalcified, the report will not be ready for some weeks. The antrum was opened from the cheek, and a mass of polypoid tissue was found in its outer part. One of the polypi, about half an inch thick, was examined bacteriologically and culture gave a small growth of the *Streptococcus longus viridans* and a few colonies of the *Staphylococcus albus*.

There was no free exudation—neither mucus nor pus. I think this explains two things: (1) that when we washed out the antrum in July the washings returned clear and gave a sterile culture, and (2) that this patient had never suffered from nasal catarrh.

This was, therefore, a chronic inflammatory infiltration of the mucous membrane, with polypoid outgrowths, but no exudation, and such a condition can evidently defy all the diagnostic tests in ordinary use. More curiously still, even an intranasal operation, in which the greater part of the middle turbinate was removed and the antrum and ethmoidal cells were opened, left the operator still in doubt as to the existence of a sinusitis which had, nevertheless, given the patient eighteen months' pain on the right side of her head, sometimes almost intolerable, and seven months' serious blindness of her right eye before we could arrive at the correct diagnosis.

Such a case must make us feel that we need some more conclusive diagnostic test of the presence of a latent sinusitis, because we obviously cannot at once proceed to an extranasal operation in every doubtful case.

On the other hand, this case suggests that an extranasal operation in some of our 30% of cases of retrobulbar neuritis of unexplained origin might help us to reduce that percentage.

Parinaud's Conjunctivitis (?). Case for Diagnosis.—O. GAYER MORGAN, F.R.C.S.

F. P., male, aged 17. Two weeks previously noticed swelling of the right pre-auricular gland and irritation in the eye. Now the lower fornix is filled with large cockscomb granulations with a patch showing some necrosis. Discrete nodules are present on the conjunctiva of the upper lid, and the superior tarsus. The cornea is perfectly bright. Cultures taken several times have been returned negative.

Mr. WILLIAMSON NOBLE said he had had a similar case in a patient of the same age and the condition had definitely proved to be tuberculous.

Colloid Bodies in the Choroid. (?) Macular Choroiditis.—HUMPHREY NEAME, F.R.C.S.

Patient, male, aged 24. The history is that within the last few months the vision of the left eye had been found to be defective when he had his eyes tested for glasses. I only examined him three weeks ago, and I found vision in the right eye was $\frac{6}{9}$ with correction of 0·25 dioptre of astigmatism. The left eye appeared to be emmetropic, and had vision only $\frac{6}{36}$ one letter, and the vision is the same to-day. I could find no vitreous floating opacities, and I thought there was no active inflammation. I could only fall back on the presumption that there were colloid bodies in the choroid. I suggest that the low vision in the left eye may be congenital, i.e., that this may be a congenitally amblyopic eye. If the condition is Tay's choroiditis, vision may be lowered if a colloid body of some size is pushing from behind into the middle of the fovea. Noticing the size of colloid bodies in microscopic sections, one sees that the nutrition of the fovea might be seriously interfered with by a single colloid body pushing forwards in this way.

Discussion.—Mr. O. GAYER MORGAN said he thought the condition was more like an active choroido-retinitis. Vessels were raised over some of the nodules.

Mr. HUMPHREY NEAME, in reply, said he did not think the raising of the retinal vessels was against the diagnosis of colloid bodies, because in sections of colloid bodies of some magnitude, the retina was pushed forward by them.

Mr. MALCOLM HEPBURN said that from the point of view of prognosis, it was very important to arrive at a diagnosis in this case. The condition did not seem to be a colloid degeneration of the membrane of Bruch, which was what was meant by the term "Tay's choroiditis." The typical appearance in that condition was of little round yellowish-white areas surrounded by an even border of pigment. Pathologically, one saw the retinal pigment pushed aside by these colloid bodies. Often they varied in size, but they retained this typical appearance. In the present case there was no appearance like that. There was much more pigment migration, especially in the left eye, and the retina was not pushed forward, so far as he could see. This appeared to him to be a case of retinal degeneration in the macular region, produced by some disease of the chorio-capillaries; in that case, unless a cause for the vascular trouble could be found and successfully treated, the prognosis was very bad. He regarded the condition in the right eye as an early stage of that in the left. The prognosis of Tay's choroiditis was very good, and vision was seldom affected, even though the retina might be pushed forwards.

Osteoma of the Right Orbit.—J. H. BEAUMONT, M.B. (for Mr. M. L. HINE).

Mrs. A. B., aged 54, sent by Dr. Gittings to the Royal Westminster Ophthalmic Hospital for investigation by Mr. Hine. The right eye has been proptosed for seven years, and it was discovered accidentally that this eye was blind. The patient has had no pain, but has had severe headaches for the last six months, and has had three epileptic fits since July last.

There is a large swelling in the right temporal fossa and over the root of the zygoma. The right eye is proptosed and diverges 45 degrees. There is loss of movement upwards and also to the left beyond the middle line.

Right vision: Shadows. Left vision: $\frac{6}{18}$; with glasses $\frac{6}{6}$. The right pupil is smaller than the left. It does not react to light, but reacts consensually. The right fundus shows a primary optic atrophy. There are scattered pigment spots in both fundi, mostly below the discs.

Wassermann reaction negative. Dr. W. J. Adie reports that there are no extra-ocular signs. Skiagrams show a dense mass of opaque tissue at the outer part of the right orbit posteriorly. There is no apparent sinus involvement.

Epibulbar Papilloma of Conjunctiva. Report on case previously shown.[1]—ARTHUR GRIFFITH, F.R.C.S.

A microscopic section was made by Mr. Charles Yow, and the report is as follows: The tumour is a papilloma of the conjunctiva. As it approaches the limbus corneæ it is seen to become shallower until it merges in the corneal epithelium. The cornea is not invaded.

Unusual Coloration of the Sclerotics.—R. FOSTER MOORE, F.R.C.S.

The subject of these notes is a woman aged 54, a patient of Dr. G. A. Arthur, who was admitted to St. Bartholomew's Hospital under the care of Dr. Langdon Brown on account of rheumatoid arthritis, for which she had been treated for some years. Both the Wassermann and the Sigma test 6·6 units were positive. Her blood uric acid content was 3 mgm. per 100 c.c.

Dr. A. C. Roxburgh was consulted with respect to the alopecia from which she suffered, but he did not consider it was due to syphilis. Mr. Frank Rose did not find any sufficient cause in her tonsils to advise their removal.

The particular ophthalmological interest is the unusual nature of the blue coloration of the eyes, which in my experience is unique.

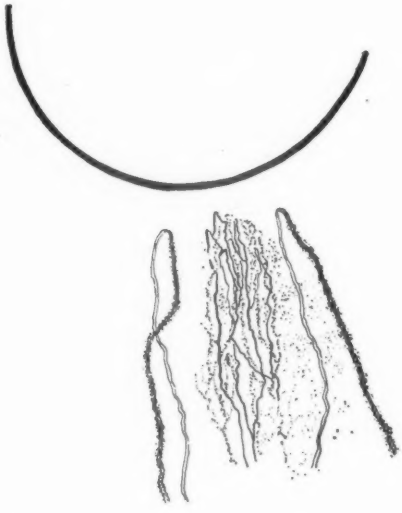
One's first impression was that she was one of the well-recognized group of cases of "blue sclerotics." On questioning her, however, she had never had a bone broken, she knew of no similar condition in any of her family or her relatives, she was not deaf, and on looking more closely it was soon apparent that she did not belong

1 *Proceedings*, 1930, xxiii, 618 (Sect. Ophth. 40).

to this group, for, as will be seen in the accompanying plate, although the colour was similar to that seen in " blue sclerotics " it was not limited to this coat, but was well seen in the plica semilunaris, and also formed a very fine blue line at the inner border of the intermarginal space of the lower lid. The colour gradually became less dense, as it was traced towards the equator, but showed no tendency to be limited to the interpalpebral area, nor did it affect the palpebral conjunctiva except in the parts referred to previously. The cornea had a faint hazy appearance. Both eyes were similar in all essentials.

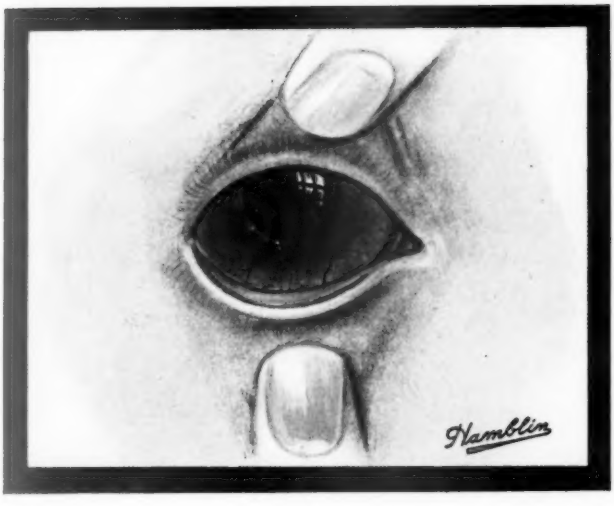
The patient was rather vague as to the length of time the condition had been present, perhaps because its development had been gradual. She however believed it had existed for something like twenty years. Dr. Arthur suggested that she had had it since childhood.

She was a myope of 4 dioptres and with correction her acuity was $\frac{6}{9}$ in the right eye and $\frac{6}{12}$ in the left. The condition of the cornea was responsible for this degree of lowered acuity. There was no evidence of intra-ocular involvement of any sort.



One would often see two fine vessels taking a parallel course close alongside each other (see fig.). One of these was mauve in colour, and the other free from all abnormal coloration.

Slit-lamp Examination.—On examination with the slit-lamp it was seen in the first place that this coloration had an entirely different cause from that of the usual blue sclerotics in that it was due to the presence of innumerable fine dark dots which could just be resolved into discrete particles by means of the instrument. The individual particles appeared to be of uniform size and of a bluish colour. They clearly were not related to the normal pigment of the eye. No suggestion of crystalline structure could be detected. They were in part scattered indiscriminately in the substance of the conjunctiva and subconjunctival tissues, but the striking feature was their relationship to the conjunctival and episcleral vessels. Large numbers of the vessels were so sheathed by the dots as to have a mauve or almost purple colour and this was true of some large vessels as well as of the very smallest ones, whilst many, perhaps a majority, were entirely free of any pigmentation.



FOSTER MOORE: Unusual Coloration of the Sclerotics.

The colour of the blood-stream in the affected vessels appeared to be of a brownish colour instead of the normal red. I imagine this was the result of the combination of the red colour of the contents of the vessel with the ensheathing dots.

I have since examined an ordinary case of blue sclerotics with the slit-lamp and have made certain that no such particles are to be seen.

G. A. Harrison has shown (*Brit. Journ. of Dermat. and Syph.*, 1924, xxxvi, 105) that in generalized argyrosis the silver is deposited in a finely granular form in the basement membrane of the sweat-glands and sweat-ducts, and here and there also in the sebaceous glands, and, later, may be deposited around the elastic fibres which lie below the epidermis. He makes no mention of a close relationship to vessels as is present here.

The Cornea.—To the naked eye the cornea appeared to be a little lacking in transparency, but there was no sort of localized opacity: the appearance was entirely homogeneous.

With the slit-lamp it was found to be due to a change in the endothelium which was uniform all over both corneæ, and which produced a fine greyness having a sort of aquatint texture.

Cause.—With regard to the cause of the condition, quite clearly this is not a case of blue sclerotics, or of ochronosis, and with these excluded, one is at first inclined to the possibility of its being due to drugs, whether taken internally or applied locally. With regard to the former, the patient had taken a number of medicines at different times; recently she had been taking a good deal of iodide of potash, and thirty years ago had a prolonged course of iron and arsenic. The condition was of many years' duration and in any case it seems unlikely that drugs taken internally could be blamed for so purely local a manifestation.

One then has to consider the possibility of it being due to local application, and as silver seems much the likeliest drug to be used in this way, and as I believe Dr. Parkes Weber thinks it is certainly due to this cause, I may be excused for considering this possibility in some detail.

We need not lay too much stress on the entire absence of any history of such application in a woman of her age, but it is pertinent to point out that there was no scarring of the conjunctiva or cornea, and no residue of any condition for which local silver treatment is usually considered appropriate.

With regard to the colour: This was almost exactly that seen in blue sclerotics and was quite unlike the café-au-lait colour which is usual in argyrosis. C. M. Myers (*Amer. Journ. of Syph.*, 1923, vii, January), in speaking of the colour of the staining, states that the silver is converted into silver chloride in the conjunctiva and that on exposure to light this becomes light-brown, olive-brown and eventually greyish-black. The brown colour he suggests is either an argentous chloride or an oxychloride, and with regard to the ultimate greyish-black he is not sure whether this is oxide of silver or metallic silver. It is usually put down to the latter. Myers has noticed that the corneal staining persisted longer in the brownish stage.

We are all familiar with cases of argyrosis of many years' standing which have been of the café-au-lait tint and in spite of Myers' interesting remarks I am not aware that I have ever seen the greyish-black colour, however old. I have seen intense and widespread staining in a workman whose duty for many years had been the preparation of solid silver nitrate. In his case the conjunctiva and cornea, as well as the exposed parts of the face and neck and hands, were all of a dull brown café-au-lait colour, and were not of the bluish colour seen in this woman.

With respect to the distribution this seems to me perhaps the weightiest evidence against a local application of silver as the cause. The coloration was quite evenly distributed all round the cornea, gradually becoming paler towards the more peripheral parts. It was not more marked in the inferior fornix than elsewhere and the lower palpebral conjunctiva was not involved.

In argyrosis due to local application I believe the inferior fornix is always more affected, and that the palpebral conjunctiva is not spared. I have never seen the colour distributed evenly around the cornea, densest where it adjoined the cornea and fading off gradually towards the periphery. I have examined a case of local argyrosis of the café-au-lait colour of fifteen years' standing by means of the slit-lamp, and the appearances were in no way similar to those in the present case. The coloration could be seen to be due to an exceedingly fine dust-like brown colour, apparently in the conjunctiva and having no sort of arrangement as regards the vessels.

Conclusion.—For the foregoing reasons it seems to me that silver may be excluded as the cause of the coloration, unless it is thought possible that if it were applied in some unusual manner the present result might ensue. Nor do I think it at all likely that the prolonged course of iron and arsenic is responsible, for it is such a common line of treatment that did any such sequel occur, we should have become acquainted with it.

I find myself at a loss as to the cause and shall be grateful to have it elucidated.

Discussion.—Dr. F. PARKES WEBER referred to a case of generalized but very slight argyrosis from the internal use of pills containing silver nitrate,[1] to illustrate how difficult it might sometimes be to ascertain the source. In the present case he thought that argyrosis from the internal use of silver nitrate could be excluded. Nevertheless, he believed the case must be one of local argyrosis, due to the external application of silver nitrate in some way or other. The remarkable dusky-blue coloration was evidently the result of the granular deposition which was seen to be specially localized about the minute blood-vessels, apparently the "venous" limbs of the capillary loops. "Dusky-blue" eyeballs (an exact description for the present case) had been known to result from silver nitrate instillation. The free borders of the eyelids had been said to be favourite sites for a line of coloration (again reminding one of the present case). In argyrosis of the eyes the silver deposit was known to be granular, and especially in the connective tissue portions, rich in blood-vessels, and not in the retina or optic nerve.

By "biopsy" examination and micro-chemical methods it would probably be possible to settle the nature of the pigment deposit, but he would not advise that that should be done.

Mr. WILLIAMSON-NOBLE said he had had a case which might have some bearing on the subject. An aged actress came and said she had not used anything for her eyelashes for fifty years except silver nitrate, and the result of that was to produce in the conjunctiva a coloration such as Dr. Parkes Weber had described; it was the colour of a dark willow-pattern plate. It was not a brownish-black, but bluish, and that supported the hypothesis that the cause was silver. He wondered whether Mr. Foster Moore's patient had been applying silver nitrate to darken the lashes.

Mr. E. R. HART said he thought the coloration shown in Mr. Foster Moore's case was similar to that seen in cartilages in the condition known as ochronosis. He remembered a case at his hospital, that of a woman who came with a chronic ulcer of the leg, and had this typical blue coloration in the ear cartilages. This made him think that in the present case the cause might be chronic carbolic acid poisoning. The patient in his case had been using carbolic oil to allay the irritation caused by the ulcer.

Mr. FOSTER MOORE (in reply) said he thought it was agreed that the coloration in this case could not be due to the internal administration of any drug. The case in which Dr. Harrison had found silver in the basement membrane of the sweat and sebaceous glands was that of a man who had had trephining performed for fracture of the skull, and a silver plate inserted which was removed fifteen years afterwards. He could not say that in the present case the granules were especially distributed along the venous loops. In the case which he had examined in which there was argyrosis of fifteen years' standing there was a sort of exceedingly fine brownish powdery deposit scattered more or less evenly throughout the conjunctiva. There was no distribution along the vessels and the separate particles were

[1] F. Parkes Weber and R. H. Norman, *Proc. Roy. Soc. Med.* (Sect. Derm.), 1910, iii, p. 75.

very much finer than in the present case. He would have thought that the coloration from local silver application was brownish, was more marked in the inferior fornix and was not limited to the ocular conjunctiva as in this case. Mr. Williamson Noble's suggestion was that silver might have been applied to the eyelashes to make them black. He had not thought of this possibility. He had wondered whether silver could have been applied in some unusual manner, but even so he was not sure that this would overcome the difficulty of the staining being diffuse all over the globe and yet escaping the palpebral conjunctiva. He agreed with Dr. Parkes Weber and he confessed that if the condition was not due to some form of local application he had no more likely suggestion to put forward. With regard to Dr. Hart's suggestion, he believed that the coloration of ochronosis was always a burnt sienna kind of tint and so far as the eye was concerned, was most marked in the palpebral fissure.

The Red Field and Optic Disc Resistance in Glaucoma and Allied Conditions.

By Ransom Pickard, C.B., C.M.G., M.S.

The object of the work embodied in this paper was to study the red field in glaucoma and any allied condition, and the factor of resistance of the nerve head to the intra-ocular pressure. Although no definite rules can be laid down as a result, the findings seem worth recording.

The work has been spread over four years. Except for a casual reference the cases in the 1925 paper (*a*) are not included. As far as possible the cases are a complete series; but certain cases had to be excluded, some because the patients were not sufficiently reliable observers, others because time prevented a full examination of the red field, another group because the cases were so advanced that any colour fields were so small as to be within the allowance for errors of observation and fixation. Some reference will be made to cases of this advanced type not included in the charts.

Colour.—Red $\frac{10}{1000}$ was chosen for comparison with white $\frac{3}{1000}$ (Traquair's discs) because red is more easily identified than green at the margin of the field; moreover, if the observer at first sees the red as pink, one may be certain that he is seeing some red at that time; whereas, if he sees the green as blue, there is no certainty that he is seeing green, so that an error might creep in because of his doubt as to the colour really seen.

Field.—The size of the field was estimated by adding the angles subtended by it along the eight principal radii, dividing the total of these by eight, the result (the average radius of the field) being used as a standard of comparison. The diameter of the blind spot or other scotoma was subtracted from the radius or radii in which it appeared.

Cups.—In my paper published in 1923 (*b*) it was shown that it was exceptional in normal discs to have a cup area of over 40%; in 127 cases between 55 and 85 years only two were more than that size. In the present series the numbers were:—

Table A.

	40% or under	Over 40%	Totals
Tension < 27	3 ...	32 ...	35
,, 27 or > 27 ...	5 ...	33 ...	38
Intermittent glaucoma ...	1 ...	6 ...	7
	9	71	80

The three cups of 12%, 23%, and 40% respectively, in the class of tension < 27 were included because the companion disc cup was large and both fields were affected. Thus, of 80 cups in this series, only nine were 40% or under, in six of which raised

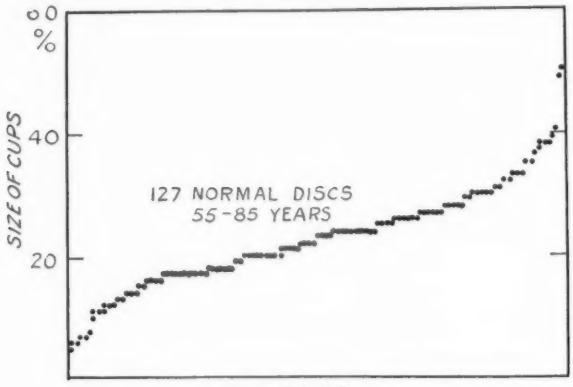


CHART I (ref. *b*).

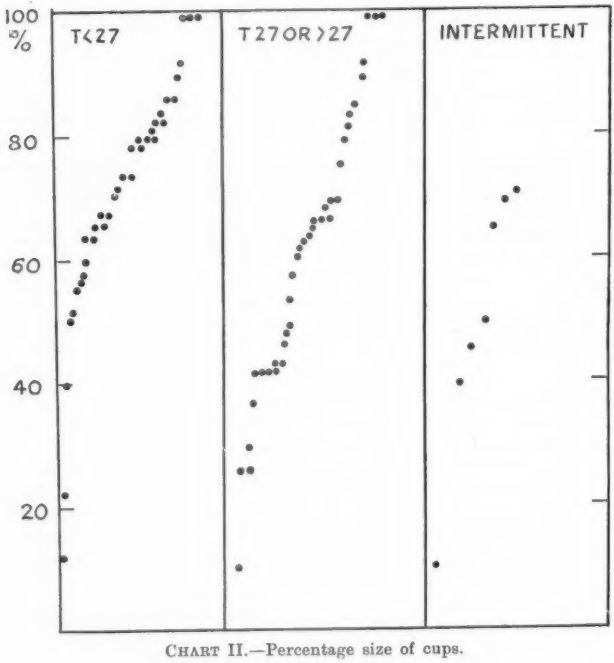


CHART II.—Percentage size of cups.

tension was found at one time or another. It will be seen that the series had as a whole, exceptionally large cups; nevertheless, the cups below 70% might be passed by many as "physiological," a most indefinite term when applied to anything which

may be expressed in a percentage with a fair degree of accuracy. That these should not be so regarded will be shown later.

Tension was estimated by the Schiötz tonometer, though as a routine it was also tested by the fingers. There are many sources of error by both methods, but the tonometer does eliminate those introduced by the lids and the tension on these by the palpebral ligaments.

Scale.—The scale used was as follows for 5·5 grm. weight:—

2°	= 30 mm. Hg	5°	= 18 mm. Hg
3°	= 25 ,, ,,	6°	= 15 ,, ,,
4°	= 21 ,, ,,		

The number of patients dealt with in this paper is 57. They are distributed as follows:—

TABLE B.

Age	Tension below 27	Intermittent glaucoma	Tension 27 or more
Below 55 years ...	4	0	6
55 to 64 ,, (inclusive)	5	3	9
65 to 74 ,, ,,	9	3	11
75 and above	3	1	3
Total persons ...	21	7	29

These figures require no comment; the bulk of the cases lie in the twenty years between 55 and 74 years (inclusive).

TABLE C.

Sex	Tension below 27	Intermittent glaucoma	Tension 27 or more
Male ...	9	3	11
Female ...	12	4	18
	21	7	29

The preponderance of the female sex is obvious in all three groups.

It will be seen that of the 57 patients, only 80 eyes are included in this paper. The reasons for excluding 32 eyes are as follows:—

TABLE D.

	Tension < 27	Intermittent glaucoma	Tension 27 or > 27
(1) Both eyes in paper	15	0	9
(2) One eye too advanced for use in charts	4	6	15
(3) One eye normal	1	0	3
(4) Other causes	1	1	2
	21	7	29

In no case in the T < 27 group was the other eye found to have a T of 27 or more, though in the four cases under (2) they were so advanced that the field was too small to make deductions as to the percentage of the red field valid. Of the other two groups taken together, 36 patients had 65 glaucomatous eyes, 21 so far advanced in glaucoma that they could not be used in this paper. Thus the grouping by tension employed is justified, inasmuch as the groups do not overlap.

Classification.—The intermittent glaucoma group has been kept intact. The remaining cases are each divided into two groups according to tension and the size of the average radius for white $\frac{3}{1000}$.

(I) Demonstrated tension of 27 or upwards. (*a*) White radius 20° or more. (*b*) White radius less than 20°.

(II) Demonstrated tension of less than 27. (*c*) White radius of 20° or more. (*d*) White radius less than 20°.

(III) Intermittent glaucoma, in which the tension has, on at least one occasion, been 27 or higher, but was usually below that point.

(IV) Advanced cases not in charts.

With one exception, all cases in the II (T < 27) group had no mists, rainbows, or pain. That case was included because it was examined over five months without a rise of tension, and the headaches had no apparent relation to the eye condition. Other cases which presented these symptoms were assumed to have intermittent glaucoma and passed to that class.

It is quite open for anyone to assert that it has not been proved that the "cases of T < 27" never had the T above 27. This is true; only by taking the tension several times during the twenty-four hours for several weeks could it be asserted with absolute accuracy that the tension was never raised—and this is, in practice, impossible. But in two advanced cases of this group I have been able to measure at such varying hours as 8 a.m. and 8 p.m. on several days in a week, always to find the tension under 27.

To those who would still regard such cases as intermittent glaucoma, the increase of pressure happening at undetected times, it must be urged that such cases must be subject to less duration of pressure, over equal intervals of time, than those in which the tension is always raised.

It having been shown (Chart II) that in these cases of low tension there is enlargement of the disc cups comparable to that in the cases of definitely raised tension, it follows that the resistance of the nerve head is less in the former than in the glaucomatous group, which is one of the points to be proved in this paper. Into the cause of this weakness it is not intended now to enter, but in a previous paper (*a*) it was suggested that the condition was "cavernous atrophy," as described by some Continental writers.

Subdivision of Groups.—Keeping the intermittent group entire, it was found convenient to divide the other two groups each into two sections, those of average white radius of 20° or more, those of less than 20°, the latter obviously comprising the more serious cases. The reason lies in the errors of the patient. Any delay in answering on his part will obviously produce the greater percentage effect, the smaller the field, which is thus less dependable in these advanced cases. Thus in the sections of less than 20° several have a red percentage of more than 100, which does not occur in the larger fields. In three of these the white field radius was 9°, 6°, and 3° respectively.

Normal Fields.—An examination of normal cases was made in order to ascertain the variations of the $\frac{8}{1000}$ white and $\frac{10}{1000}$ red fields. It was found that of 19 eyes, six showed a red percentage of less than 60, most of the remainder being above 80%: i.e., $\frac{1}{3}$ showed small fields. No cause was found for this; all these cases showed normally sized white fields, nor were there widely varying conditions of illumination such as might account for this variation.

Graphs of Red Fields.—It is this group of small red fields in normal cases which prevents any deduction in an individual case as to the abnormality of the red field; only by employing graphs of a series of cases can any tendency to diminution of the red field be demonstrated.

Chart III.

Two Groups over 20° White Radius.—In the chart showing the red percentage in cases of over 20° average white radius, the close correspondence between the two groups is at once seen, so that either might be substituted for the other without any great inaccuracy. Only for the larger values can it be said the glaucoma group has a slightly higher range than the group in which increased tension was not found. In the first quarter the two groups are smaller than the normal by an insignificant amount, in the middle two quarters both groups are smaller than the normal by a little more than 20°, in the last quarter the glaucoma group approximates to the normal, the other group remaining somewhat less. Taken as wholes, it may be said that about half of each group has a distinctly smaller red field than the normal.

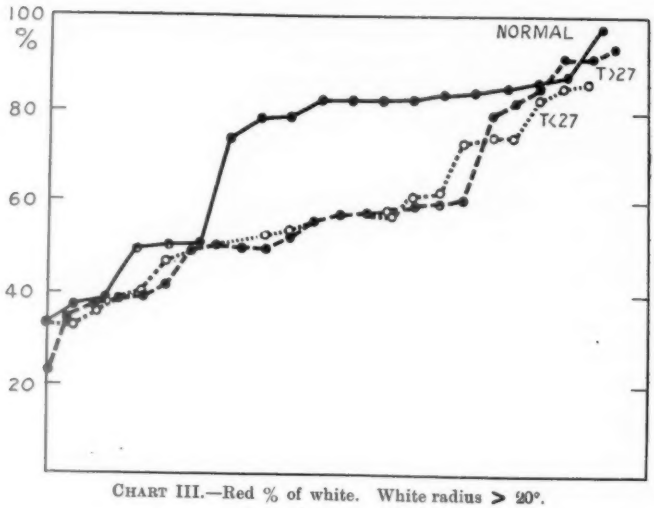


CHART III.—Red % of white. White radius > 20°.

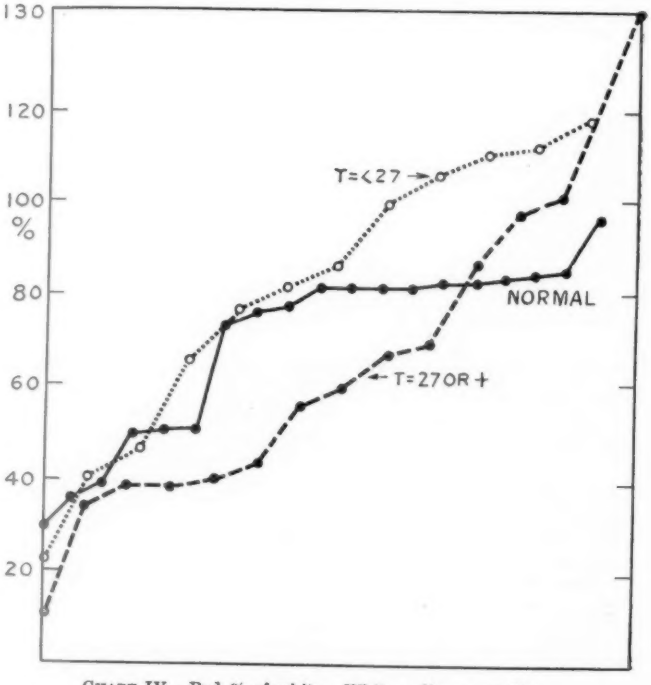


CHART IV.—Red % of white. White radius = < 20°.

Chart IV.

Two Groups under 20°.—It will be seen that of these two groups the glaucoma group approximates towards the glaucoma group of 20° or over, except for three of more than 100%, while the lower tension group shows a higher percentage of red field even than the normal group. This may be due to errors of observation, but it may be safely said that in the smaller fields the red is retained to a higher degree than in the larger: i.e., while the peripheral red field is lost easily, the central is retained tenaciously.

Chart V.

Intermittent.—If it be assumed, notwithstanding the small number in the intermittent group, that it is a good average representation of such cases, a base line can be taken approximately equal to that for the previous two groups. Then it will be seen that the graph of this group approximates to that of the normal group. This would be according to expectation, for since in this class there is only an occasional deviation from normal pressure, there should not be any great variation from the normal in the red field.

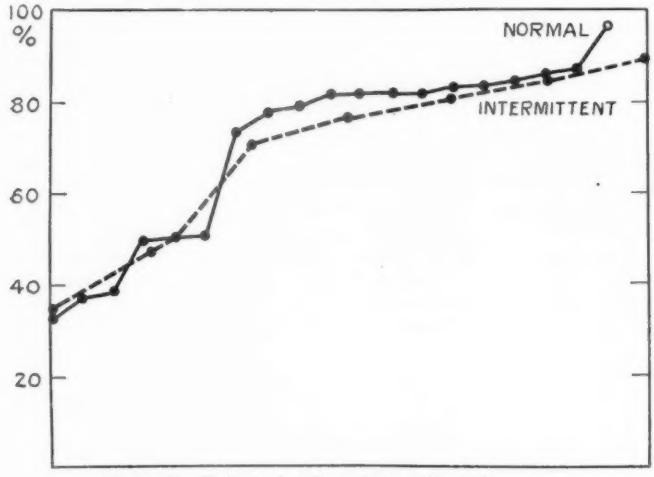


CHART V.—Red % of white. Intermittent glaucoma.

Thus the main anomaly in the red field graphs is that, while the two groups of T over and under 27 have a fairly similar difference from the normal, the intermittent class approximates to the normal. There would appear to be some difference in causation to account for this inconsistency. If it be assumed that in the group of T < 27 an abnormal weakness in the nerve head causes this to give way easily, while in the groups of intermittent glaucoma and of T 27 or higher there is an average resistance power, then all the groups come into line; each behaving according to the interaction of two factors, the resistance of the nerve head and the tension of the globe.

It is not maintained that in the T < 27 group the T is never above that amount. What is asserted is that, since repeated trials failed to show a higher T than 27, the nerve head has given way under a less total exposure to pressure than in the other groups. As will be attempted to be shown later, in some cases of this group reduced T after operation has not prevented further downhill progress in the case.

Chart VI.

Spot Charts.—The actual sizes of the red and white fields radii are shown by the spot chart. In these the oblique line should be touched by cases in which the red percentage is 100, above this line the red is less than 100, below it more than 100%. The higher the spots the larger the white field. The charts may be tabulated thus :—

TABLE E.

Size of white field $\frac{3}{1000}$ *(radius).*

	Normal	Tension < 27	Tension 27 or +	Intermittent
40°-31° ...	16 ...	9 ...	8 ...	3
30°-21° ...	3 ...	15 ...	13 ...	7
20°-11° ...	— ...	9 ...	11 ...	1
10°- 0° ...	— ...	3 ...	5 ...	—

The close resemblance between the T < 27 and T 27 or more is obvious. The intermittent class is midway between these and the normal class in the size of the white $\frac{3}{1000}$ radius.

Clinical Progress of T < 27 Group.—Eight eyes definitely became worse. Three of these (comprising two patients) were trephined but the fields continued to get small and the cups to increase. It is interesting that the untrephined eye in one of these two patients—too advanced to be included in the charts, too bad to make operation worth while—never showed a rise of tension.

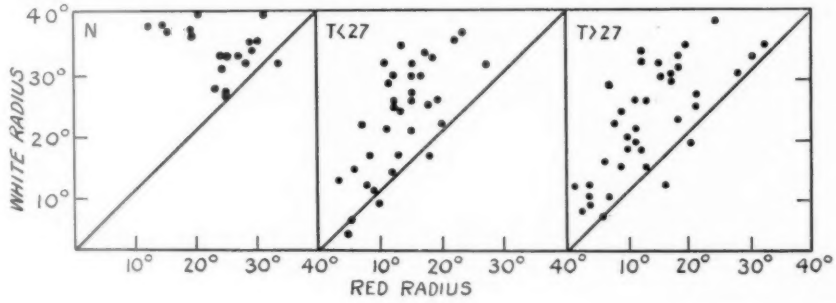


CHART VI.

Eight eyes had an increase in the size of the cups but no decrease in the $\frac{3}{1000}$ field, though three of these had a decrease in the $\frac{1}{1000}$ field.

Eleven eyes were definitely no worse in cups or fields over a period varying from eight months to three years and nine months. In these cases the $\frac{3}{1000}$ fields varied from 32° to 9°, the cups from 100% to 70%, with one exceptional cup of 12%.

The remaining eyes have no records long enough to give any estimate as to their course.

The outlook in these cases is so varied that no generalization can be made. Speaking roughly, the larger the cup, the smaller the field and the worse the prognosis : but a stationary case observed for three years and nine months, with a cup 100% and 4 D deep, a white field of 32° radius for $\frac{3}{1000}$, 13° for $\frac{1}{1000}$, and a tension of 18° forms a remarkable exception. It is considered to be an arrested case.

If it be assumed that the process or processes causing weakness of the nerve head are of varying intensities and durations, so that some cases come to an end before the field is greatly affected, while others progress much further, then the varying clinical pictures are explained. If it be also assumed that the nerve fibres and connective tissue elements in the nerve head are variously affected, the very

severe course of the trephined cases may be explained by an affection of the nerve fibres, besides that caused by the normal eye tension upon nerve fibres insufficiently supported by surrounding connective tissue.

Advanced Cases T < 27 (not included in Charts).—In one eye the red field was not taken, so this eye could not be included in the charts. In the other three eyes the fields were too small to be usefully included, an average radius of 3° or less. In these the $\frac{10}{1000}$ red field was about the same size as the white $\frac{3}{1000}$, the cups being 100, 90 and 70%.

An advanced case of glaucoma with both eyes affected is now to be mentioned.

H. H., 58 years.

		Tension	Cup	Vision	White field $\frac{3}{1000}$
October 4, 1920	Right eye	+	48%	$\frac{6}{9}$	23
October 11, 1920	—	Trephined			
January 22, 1925	—	18	66%	$\frac{6}{18}$	18
October 4, 1920	Left eye	+	57%	$\frac{6}{6}$ (4)	33
January 23, 1922	—	Trephined			
January 22, 1925	—	25	76%	$\frac{6}{6}$ (1)	12
November 8, 1928	...	Reported to see very little. Cannot distinguish red but does other colours.			

The loss in the right field in 1920 was sectoral with a large Ronne's step. The loss increased and coalesced with an enlarged blind spot.

In the left eye the loss began in 1922 as a paracentral annular scotoma nearly confluent with the blind spot, the outline of the field being nearly full.

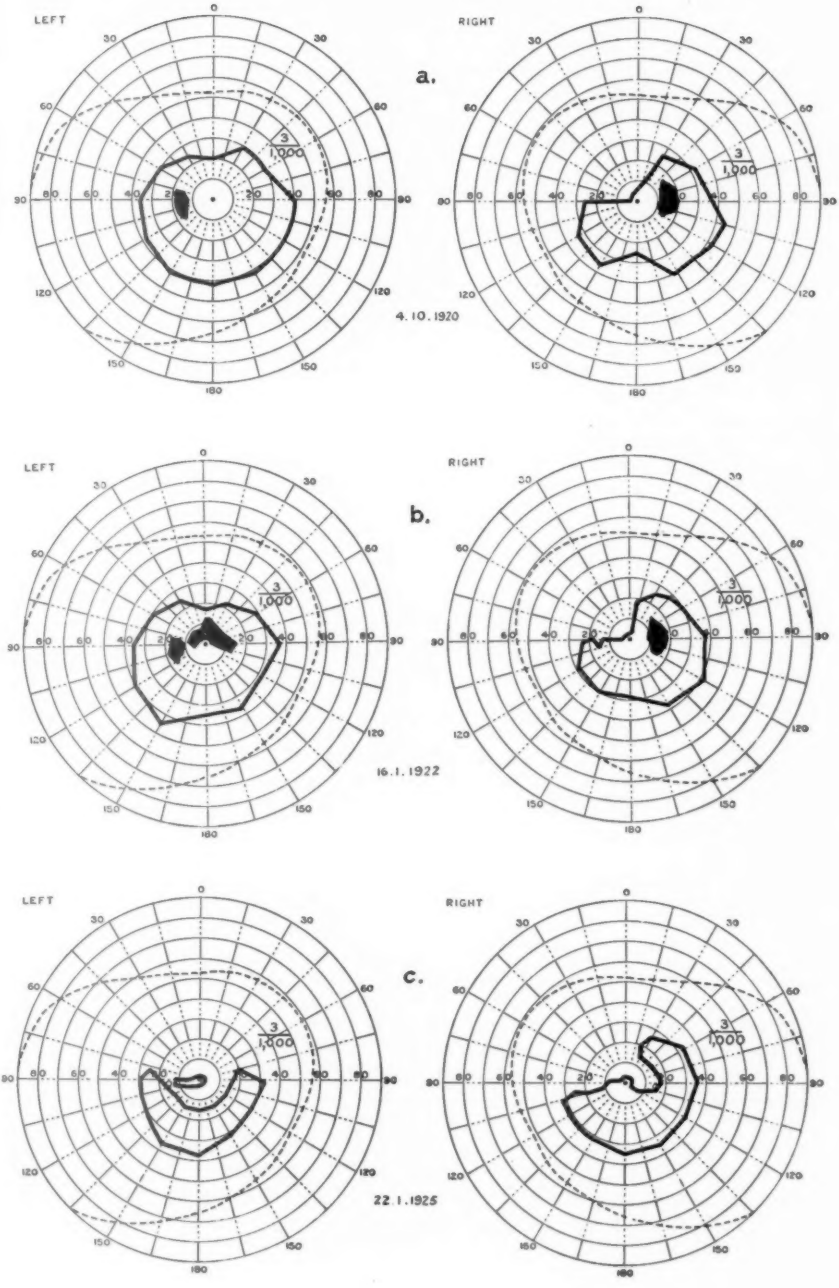
This scotoma enlarged, the upper half of the field disappeared, and the blind spot enlarged into a wide annular scotoma of 180°, which separated the central and peripheral portions of the field.

This is a case in which glaucoma existed in combination with a weakness of the nerve head; so that, although pressure was relieved, the loss of the field progressed, and but little sight is left.

The belief that cases of undemonstrated raised tension and large cups must have raised tension at some unobserved times is founded upon variations actually demonstrated at different times in some cases. But this generalization leaves out the possibility of inherent or acquired weakness of the nerve head, and means the denial of the variation of one of the two factors which together give the reason for the cup enlargement, when one of them—the ocular tension—is known to vary. This position is unreasonable on grounds of probability, and it is hampering to research. For it is by sorting out cases which agree in some signs but differ in others that an appreciation of the varying factors at work can be made. The continued downward progress of some cases of operated glaucoma, when no such fresh factor, such as opacities of the media or inflammation, has been introduced, suggests a missing influence. The varying resistance of the nerve head supplies this.

Whether it is an inherent weakness in the sense that the nerve head tissues are in some sense congenitally unable even to withstand normal pressure after middle life, or whether it is a pathological condition, cannot be decided without a microscopical examination—perhaps one cause in some, the other in other cases is at work. The advanced cases, especially those which progress after trephining, suggest an actual contraction rather than a mere recession, a contraction following upon a pathological alteration.

Conclusions.—(1) In intermittent glaucoma, as far as can be judged from seven cases, the red field approximates to the normal. Both the groups of T above and below 27 show a curve 20° smaller than the normal in the middle two-fourths for cases having over 20° white field ($\frac{3}{1000}$). In both groups of under 20° white ($\frac{3}{1000}$) the central red field is retained tenaciously, the low T cases having a larger red field than the high T group.


LEFT
RIGHT
a.
4.10.1920
b.
16.1.1922
c.
22.1.1925
3/1,000

(2) The two groups of T above and below 27 are practically identical in age, sex, size of disc cups, degree of loss of red and of white fields, but differ in tension.

(3) There being no clinical evidence of increased tension in the T < 27 group, the giving way of the disc cup must be due, at least in part, to weakness in the nerve head, perhaps in some cases, to active recession.

(4) This conclusion is supported by two cases in which trephining has not arrested the loss in the fields; i.e., in which the loss proceeds in the presence of a definitely subnormal tension.

(5) There is some evidence which suggests that the progress of some of these cases does become arrested. If this is so, it tends to prove that the main factor is the weakness in the nerve head and not the pressure in the globe.

(6) There is no evidence as to the nature of the weakness in the nerve head. None of the cases had any symptoms of general disease of the nerves or central nervous system.

REFERENCES.

PICKARD, R., (*a*) *Brit. Journ. Ophth.*, 1925, ix, 385; (*b*) *id.*, *ib.*, 1923, vii, 81.

Mr. PICKARD said he desired to make the point clear that this was not a selected group of cases which he had dealt with in the paper; they were, as far as they could be, a continuous series. He had gone to work on a definite system. He was working on the cases for four years, and it was not until the end of that time he collated his results, to prevent him being biased in any way.

Section of Orthopædics.

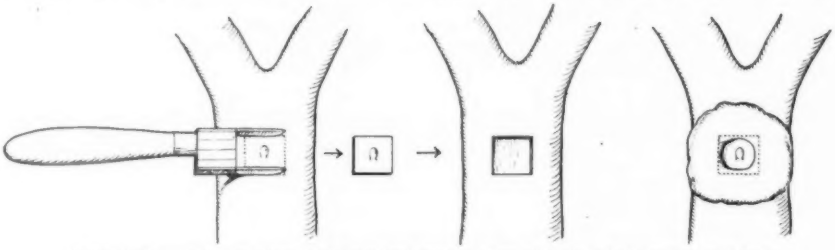
[*October* 15, 1929.]

The Various Uses of the Bone Graft.

By Fred H. Albee, M.D., F.A.C.S.

There is no branch of medicine that has not been illuminated by the study of its analogue in lower animal life and even in other kingdoms besides the animal. Comparative anatomy, physiology and pathology have grown to much more than academic importance, as they have been woven into diagnosis, therapeutics and surgery. We do not know the full debt we owe to those who have developed problems out of the dim regions of purely scientific research into the full light of practical application to human disease.

The origin of grafting, if recorded at all, is so obscure that I have not been able to trace it. I was not unfamiliar with the process in fruit-trees before I undertook to apply the principles to the surgery of bones. As might be expected, I found much in the study of plant-grafting to guide me in bone-grafting. One can safely assume that the principles of plant-grafting cannot be violated in the grafting of tissues of a higher form, and that grafting of bone cannot be as readily carried out as grafting of vegetable material. These postulates arise from the very nature of biological principles and the decrease in adaptability with increase in specialization



Method of removing graft consisting of bud with portion of bark and alburnum, and fixing it in a gutter in the host. The double-bladed knife, like the motor-driven twin saw, insures accuracy of fit. The paraffin dressing provides immobilization and access to light.

of tissue. There are three inviolable rules in plant-grafting: the tissues must be applied like to like, the contact must be most intimate, and they must be immobilized in that position. These are, at least, as important in bone-grafting. In the more highly specialized animal tissue, however, it is desirable, if not essential, that the graft be autogenous.

When the bark and bud are removed for the scion, a piece of bark of the same size and shape is removed from the host (see fig.), so that like tissues of the scion will be applied to like tissues of the host and so that immobilization will be facilitated. Interruption of the circulation of sap in the host is thus minimized. The "taking" of the graft consists of the laying down of new cells, partly by the host and partly by the scion, which eventually fuse.

It is thus evident that the cells of the scion preserve their vegetative and proliferative power. The re-establishment of the circulation is simpler in the plant, but it is obvious that the sap must permeate the cells of the scion or the latter will eventually die. In bone, re-establishment of circulation becomes a problem the solution of which governs the whole question of surgical technique.

In the plant, as in bone, like tissues are applied to like because there is a force, called by John Hunter the "stimulus of incompleteness," which tends to force

tissues to complete a broken surface, and because this force is greater when it is mutual, that is to say, when it is applied to two broken surfaces of like tissue.

Of all the layers in a tree, bark, alburnum, heart-wood and heart, the alburnum is the important one in grafting, since it is the proliferation of its cells in scion and host that brings about the union. In bone, and especially in long bones, the structure is much more complex, and all the layers, periosteum, cortex, endosteum and marrow, take part, to a greater or less extent, in union. It is for this reason that accurate, power-driven instruments are necessary for preparing graft and graft-bed ; perfect coaptation, so essential to the nourishment of the graft, and to its function as a vessel-conducting and osteogenetic unit, can be attained in no other way. If conditions were similar in the tree, power-driven tools would be necessary for grafting in the vegetable kingdom as well.

Immobilization is effected in plant grafting in two ways, in addition to the fixation provided by the accurate fit of the scion : the site of grafting is either bound with many layers of fabric or is covered with wax, or better, paraffin (see fig.). These sealing substances serve as a sterile dressing as well, since they keep out fungi. If paraffin is used, immobilization is just as secure, and light (especially the ultra-violet) is allowed to penetrate to the wounded tissues and especially to the bud, which needs the ultra-violet for the metabolic activity of its chlorophyl.

I do not know the history of the double-bladed knife (see fig.) used for plant-grafting, to ensure identity in size and shape of graft and graft-bed. I first saw it many years after I had designed the twin saw. Its use was demonstrated to me by the late Mr. W. J. Krome, in his citrus grove in Florida. Mr. Krome, after many years as a distinguished engineer, during which he supervised the construction of the Florida East Coast Railway to Key West, retired to indulge his interest in biology. Always a keen student of this subject, he used his declining years to the advantage of mankind as well as to his own great enjoyment. He made important studies of tropical and subtropical vegetation and particularly of tree-grafting. He was one of the few men who have the foresight and the qualities to arrange for the last chapter in their lives so that it will not be an anti-climax. When I last saw him he was full of his hobby and showed me his work and his methods. The twin knife was almost his whole armamentarium.

I did not design my twin saw from this twin knife, but they were equally a response to the demands of the operation. The twin saws may be regarded as cutting calipers since they ensure that the graft will fit the graft bed. In many cases they are used actually to measure distances and to record these on the bone to be cut.

Plant grafting and bone grafting thus have the same objective and are carried out according to the same fundamental principles. The application of these in bone must be more meticulous, since there are added difficulties in a relatively highly specialized tissue nourished by a system of closed vessels.

In the plant the only force to be counteracted is that of the wind, and this only when the scion is a large shoot. In bones there is the pull of muscles, both tonic and voluntary, and the exaggeration of the former by reflex from pain. Fixation and immobilization therefore present difficulties which must be met in a special way. It is necessary to consider bone, therefore, not only as a living tissue with an intricate circulation and metabolic functions, which are only beginning to be understood, but as a rigid piece of material which must be held in place when continuity is broken. For help with the mechanical problem one must go to the joiner and study his various forms of mortise and how he selects each according to the mechanical demands of the situation. Union of graft with host, however much it may be affected by mechanical exactness, is dependent on the principles of biology and physiology that govern the transplantation of tissue as a living, functioning structure. The graft lives if it is supplied with blood from the host

bony union with the host follows in the same way as it does after fracture, by the formation of a granulation-tissue bridge containing osteoblasts which determine first the deposition of soft callus and then its impregnation with lime salts. Having applied the graft in the way most likely to favour its union with the host, one must have faith in those mysterious processes by which the graft remakes its structure, alters its shape and takes on added volume and strength in order to accommodate itself to the demands of its new environment.

My first application of the bone graft was to the spine in Pott's disease. Since then it has been used in grafting most of the bones in the body and for a variety of purposes.

The Inlay Graft.—The inlay graft in long bones is a perfect example of the ploughed-and-tongued joint, as well as of the application of like tissue to like. It is, therefore, a complete response both to the biological teachings of comparative botany and physiology and to the principles of mechanics that guide the joiner.

The inlay graft is applied principally to the long bones in cases of non-union. In typical non-union the ends of the fragments are avascular, circulation being deficient for a greater or less distance back from the region of the fracture. The bone is sclerosed and eburnated and contains few, if any, osteoblasts. Its vitality, and hence its power of regeneration, is slight. Before the graft came into use it was necessary to cut away all this bone in order to reach healthy bone with greater capacity for regeneration. Now, it is possible (and naturally preferable) to carry the vitality of the healthy bone forward to the fracture surfaces, with the minimal sacrifice of bone tissue and length of limb. A bone graft inlaid into the fragment, reaching far enough back to tap the blood supply and sources of nutrition and osteogenesis in the healthy bone of the host, will bring to the zone of non-union forces which are of the same order as the reparative and osteogenetic power of recently fractured bone. The source of regeneration lies mostly in the soft-tissue structures that sheathe the bone and are enclosed in its medullary canal. Hence the graft and graft-bed are full of thickness, so that like tissue may make contact with like from marrow to periosteum. The vascular channels, especially the capillaries, in graft and host bone unite. Thus the graft obtains nourishment, not only for its own existence and subsequent growth and rearrangement, but for the nutrition of the avascular and impoverished ends of the fracture fragments. It is occasionally necessary to insert a second graft into the first one, or to use a portion of the original graft as a sliding graft when a mishap, such as infection or fracture, has occurred. In such cases one has an opportunity, sometimes late and sometimes comparatively early, of observing the condition of the original graft. It is always well vascularized and bleeds freely when cut. These surgical observations have been confirmed by Sir Arthur Keith, who injected the blood-vessels to demonstrate the vascularization of the graft. He was good enough to show me his specimens at my last visit to the Royal College of Surgeons.

One must not picture the graft as persisting in the form of a slender bridge between two fragments of sclerosed bone. The graft not only throws out callus and ossifies it, but responds to biological demands, according to Wolff's law, and enlarges and thickens and takes on the structure of the host bone. More than that, the sclerosed fragments respond to the demands of restored function. Just as disuse brought about atrophy of the organic structures (vascular and cellular) in the useless fragments, so now restoration to usefulness and subjection to stress stimulate reconstruction of the fragments. Vessels grow in, the dense bone is absorbed and the original structure is eventually restored. It is again Wolff's law, which is only one striking expression of the law governing the relation of structure and growth to function.

In the case of non-union of fracture of the patella, in which muscle tension keeps the fragments apart, it is necessary to alter the shape of the graft to suit the mechanical conditions. The graft must have some sort of a device, in the nature

of a flange, to resist the tension. I have used an H-shaped graft, one cross-beam being inlaid in each fragment, at right angles with the extremity and the line of tension. The graft-bed, of course, is made of the same shape, advantage being taken of the caliper action of the twin saws to ensure absolute correspondence of bed and graft in size and shape. The periosteum stripped back from the patella before the graft-bed is prepared, is replaced over the periosteum of the graft, and circulation to the graft is thus insured. The cancellous bone of the graft of the patella is also very vascular and contributes to the nutrition.

Grafts of irregular shape are used for loss of substance of the cranium and for repair of the mastoid as well as for cosmetic purposes, especially about the face.

Arthrodesis by Bone Graft.—In painful or progressive destructive lesions of joints, there may be no recourse but to immobilize the joint by arthrodesis. This may be done without arthrotomy by grafts from one member of the joint to the other. The method is particularly applicable to the hip. It is carried out in different ways, a bony bridge being established, by means of a graft or grafts, between some part of the ilium and the great trochanter. In some cases the graft is taken from the trochanter itself; I usually take it from the tibia or the outer table of the ilium. Two grafts are preferable because of their truss-work effect. One end of each is set in a depression of the ilium and the other in a gutter in the great trochanter. Extra-articular arthrodesis has two advantages: the lesion is not disturbed (a point of greatest importance in tuberculosis), and the grafts are embedded in normal bone capable of providing the most favourable conditions for union and the least risk of infection.

These grafts are not applied as ideally as they are in the long bones for non-union. The cortex of the graft is applied to the cancellous bone of the host. However, since the latter contains the vessels that accompany the marrow and is therefore very vascular, the graft-bed is eminently able to provide vascular connections for the severed vessels of the various layers of the graft. The vitality of the grafts is well maintained, and they increase in size and seldom fracture, especially when two are used.

The Bone Graft for Diseases of the Spine.—The application of the bone graft to the spinal column is a form of extra-articular arthrodesis. Here the tibial graft is fixed in a gutter made by splitting each spine in two and fracturing one half laterally. The graft traverses not only the affected vertebræ but two above and two below, except in the case of the lumbar vertebræ when only one above and one below receive the graft. The purpose of the graft is not only to supply immobilization, but to bring fresh circulation to the lesion itself. The graft includes all the layers, the vitality of which is ensured by the linking up of the circulation, especially of the medulla and periosteum, with that of the host. The graft unites with the split spines, and through them refreshes and reorganizes the disrupted circulation of the diseased bodies. This explanation of the success of the method has been questioned, and the full argument may not be presented here. It is possible that the beneficial effect on the lesion may be attributed purely to the mechanical effect of the graft; it not only immobilizes but also supports the bodies against the mutual pressure which adds materially to the destructive effect of the disease itself. This support is especially effective, because it is applied as a leverage as far from the fulcrum as possible, namely at the tips of the spines.

The bone graft is used for other spinal abnormalities such as scoliosis, spondylolisthesis, and compression fracture of the vertebral bodies. The method of application is similar. In scoliosis it is inlaid into the spines at the most pronounced portion of the primary curve, after the maximal amount of correction has been carried out. It is particularly advantageous in the paralytic type of scoliosis.

It is occasionally used for extra-articular arthrodesis of other joints, even the sacro-iliac.

Other Applications of the Inlay Graft.—An unusual use of the inlay graft is in

the correction of contracted pelvic outlet. I have not been in a position to carry out this procedure in more than a few cases, but its reasonableness and the success so far attained should bring it into decided favour. The procedure is the reverse of that followed in non-union of the patella. The symphysis is prized apart and a graft is inserted into the superior surfaces of the pubic bones to hold them at the required distance. The operation is scarcely more formidable than symphysiotomy or pubiotomy, and it has the striking advantages of permanent enlargement of the parturient canal and of stability of the pelvis. The graft is again an inlay from the tibia.

The graft is used to change the contour of a joint which is not carrying out its function or is allowing abnormal mobility. Habitual dislocation of the patella is due to malformation of the condylar groove, one of the margins not being pronounced enough to retain the patella in its proper alignment during its excursions as the knee moves. Half the battle is won when the cause is realized, for the cure consists of correction of the groove by bone transplants. One condyle is elevated anteriorly, and held in that position with a wedge graft.

The Onlay Graft.—The onlay graft may be used when the bones to which it is applied are normally vascular and of undiminished osteogenetic power. For instability of the foot, whether congenital or acquired, bone transplants are used, especially when tendon transplantation is not feasible or fails. Campbell uses a "bone-block" or shoulder of bone at the posterior confines of the ankle-joint, to prevent foot-drop and hold the foot in position for walking.

These grafts are onlays, and are intended not, as the inlay generally is, to replace missing structures and to restore original alignments, but to add to or accentuate existing contours in order to overcome some abnormality. An onlay graft of this type has been used to correct abnormal facial contours. I have restored a chin when the contour was so defective as to constitute a serious blemish and militate against the patient's peace of mind or ability to earn a livelihood.

Use of the Graft in Congenital Dislocation of the Hip.—A mixed type of graft is used in the surgical treatment of congenital dislocation of the hip. If the acetabulum is too shallow to hold the head of the femur in place, the rim is fractured outwards and held in position by a key-wedge from the tibia or from the outer table of the ilium. If the head cannot be returned to the acetabulum, except by inflicting such trauma to the head and acetabulum as might produce serious limitation of movement, a new acetabulum may be formed on the dorsum by turning down a shelf from the outer table over the femoral head and capsule, and bracing it by struts from the same source.

The Peg Graft.—The peg graft finds its most common use in ununited fracture of the neck of the femur. A peg shaped from a graft from the crest of the tibia, including all layers, and ½ in. in diameter, is fitted into a hole drilled from the trochanter through the neck into the capital fragment. The dowel tool which does the shaping corresponds to the drill which makes the hole, so that accuracy of fit is mathematically certain. In no type of grafting can this be dispensed with, but in the peg graft it is even more necessary since the method does not fulfil all the physiological requirements of grafting; the layers of the graft cannot, of course, be all brought into contact with similar layers of the host. The marrow, endosteum and periosteum of the graft must carry the circulation through from the periosteum of the trochanter into the zone of fracture and the capital fragment, the latter being often poorly nourished. The Haversian canals of the graft receive a certain amount of blood supply from the vascular marrow in the cancellous tissue of the host, but, since the Haversian system is arteriolar, anastomosis is not as readily established as it is in the capillary systems of the marrow, endosteum and periosteum. It is for this reason that one should always take care to leave as much as possible of these structures attached to the graft.

The peg graft may also be used in fresh fracture of the neck of the femur in specially selected cases. It has many other applications: to supplement an inlay

graft in non-union of long bones; to aid in arthrodesis of the shoulder, the astragalo-scaphoid and other joints; to fix an onlay graft, if this is used for non-union of long bones; to supplement or take the place of kangaroo tendon in the fixation of inlay or other grafts.

The Screw Graft.—The screw graft is made by machine from living autogenous bone, in the operating room. A bone peg is first made and this is then threaded. It is used instead of the peg graft when the latter will not get sufficient grip to hold the main graft or to approximate it closely enough to the host bone. When the host bone is thin, as in the cosmetic building of the chin already referred to, the screw graft must be used. It is also employed in holding the graft used in the repair of fracture of the patella, to insure that the graft will not be pulled from its bed by the tension of the quadriceps. It is preferable to the peg graft for fixing the onlay graft if this is used. It is ideal for use in fresh oblique fracture because it draws the fragments together. In ununited oblique fractures it may be used alone or as an auxiliary to the inlay graft.

The Bone-muscle Lever.—The bone-muscle lever which I employ in the reconstruction of the hip-joint is actually a process of autogenous grafting, since the bone fragment of the lever is detached from the femur in order to be set at its new angle. In this way, the part of the trochanter to which the muscles are attached is carried further from the acetabular fulcrum and the action of these muscles is thereby accentuated. Not only is the leverage action of the neck of the femur restored and the function of abduction re-established, but the reconstructed head is prevented, by the more effective tension of the muscles, from dislocating when the limb seeks the mid-line.

Less Common Uses of the Bone Graft.—In severe cases of scoliosis where the ribs impinge on the brim of the pelvis or actually invade the iliac fossa, a bone graft is used as a prop to hold the ribs up, not only to restore the ribs to a less painful position, but, indirectly, to support the spinal column in better alignment and to supplement the direct effect of the spinal graft. The graft is fixed to the brim of the pelvis and (usually) the tenth rib.

The bone graft is used in the creation of digits. After the soft parts have been constructed by the pedicle graft, the new digit is given the necessary rigidity by means of a bone graft fixed to the distal end of the bone of the stump.

I have given a sketchy description of the various uses to which the bone graft has been put, and I have outlined the general principles upon which bone grafting is based. It is true that these principles are not followed equally closely in all the uses of the bone graft which I have mentioned, but conditions are not always suited to the ideal use of a bone graft. On the other hand, certain modifications have been advised by others which depart needlessly from the first principles. Some of these I have never used; others I have experimented with under exceptional circumstances, but always to my regret. Instead of taking the time to discuss the disadvantages of these methods, I have laid down the general principles which underlie all bone grafting and have left the fallacies in other methods to inference.

It would be as contrary to the trend of medicine as it would be discouraging, to think that the possibilities of the bone graft have been exhausted. I can suggest one field still which I have scarcely more than begun to explore, and that is the replacement of congenitally absent bones. I have on numerous occasions, by manufacturing a radius, restored a considerable degree of function to a useless hand in a child. I have already referred to the treatment of congenital contracted pelvis by means of the bone graft. This field will no doubt be further explored. As the fundamental sciences throw still more light on disease and repair, the uses of the bone graft will be further developed. It is that hope, as much as the desire to ameliorate the ills of humanity, which gives zest to one's endeavours.

JOINT DISCUSSION No. 5.

Sections of Comparative Medicine and Surgery.

[*January* 22, 1930.]

DISCUSSION ON ACTINOMYCOSIS COMMON TO MAN AND ANIMALS.

Dr. L. Colebrook: In the first place it may be well to consider the use of the term "actinomycosis" in the light of present-day knowledge. If we were discussing tuberculosis we should all agree, I suppose, that the term connotes a wide variety of clinical conditions all referable to infection by Koch's bacillus. This is not the case with actinomycosis, for the term has been used in the past, and is still used, to cover infection by several microbic species, some of them widely different. The clinical manifestations of these infections have often points in common, but they are by no means identical even in this respect.

The pathological conditions included in human and veterinary medicine under the term "actinomycosis," may be described as chronic, localized, suppurative processes—or, if preferred, granulomata. We meet them usually in the form of nodules, abscesses or fistulæ; we exclude the other granulomata, tuberculosis, syphilis, glanders, leprosy, etc., by the clinical characters or by the appropriate laboratory tests, and we make a diagnosis of actinomycosis, sometimes, but not always, by the naked-eye observation that the pus contains "sulphur granules." When we attempt to classify this heterogeneous group of clinical conditions we get into difficulties at once. We cannot adopt a purely clinical classification because the clinical picture presents no unmistakable features—nothing comparable, for example, to the skin picture of erysipelas in man; nor can we, as yet, make a complete bacteriological classification in terms of the infecting organisms, because we are not sufficiently familiar with many of these to differentiate them.

The group of organisms in question is a large one, and many of them are difficult to cultivate. Their differentiation must necessarily therefore occupy a number of years. Some progress has, however, been made in this direction.

In Table I I have made an arbitrary distinction between the infections which produce *granules bearing clubs*, and those which do not. The presence of granules irrespective of their association with "clubs" is a less satisfactory basis of classification, because one cannot always be sure, by naked-eye observation alone, whether a suspected granule is really a genuine aggregated mass of bacterial growth or merely a compact aggregation of leucocytes. Vigorous shaking in water will help to decide the point when one is dealing with quite fresh pus, but a simple microscopic examination (unstained) of the suspected granule is much more reliable and ought to be resorted to in every case. That will show at a glance whether our granule is a mass of leucocytes or no—and it will also reveal the presence of clubs. The histologist will perhaps object that we should not require granules to be visible to the naked eye before we recognize actinomycosis, and technically he is right, but we are concerned here with practical surgery and with lesions large enough to call for treatment.

TABLE I.

The term "actinomycosis" may refer to:—

I.—*Infections associated with granules bearing clubs*, and due to:

(1) *Actinomyces bovis*, anaerobic type [Wolff-Israel].
In man: jaw, thorax, abdomen.
In cattle: jaw, udder.

(2) Mycelial organisms distinct from *A. bovis*.
In man: "madura feet" and miscellaneous cases [Wynn, Cohn, etc.].
In cattle: (?).

(3) Actinobacillus of Lignières and Spitz.
In man: one case reported; (?) others.
In cattle: "woody tongue" and skin lesions.

(4) Staphylococcus.
In man: (?).
In cattle: udder (cow and pig).

(5) *Bacillus pyogenes*.
In cattle: lesions of jaw, etc.

II.—*Infections by mycelial organisms, but showing either no granules or granules not bearing clubs*, due to:

(6) Unclassified organisms (streptothrix).
In man: miscellaneous cases, reported by Birt and Leishman, Henrici and Gardner, etc.
In cattle: miscellaneous cases, e.g., those of Siberschmidt, Nocard, and others.

First among the conditions associated with the formation of granules bearing clubs (groups 1—5 of Table I) I have placed the infections by the well-defined anaerobic organism *Actinomyces bovis*, described by Wolff and Israel [1], Homer Wright [2], Colebrook [3] and others. There has been a steady growth of bacteriological opinion during the past twenty-five years that this species is responsible for most of the cases of human infection associated with granules and clubs. The great majority of such infections in man are found in one of three situations, viz., about the jaw and neck, the thorax and the abdomen—particularly in the right iliac fossa. I have no first-hand experience of the cattle infections by this organism, but I understand that the lesions are most commonly found about the jaw and the udder.

Second in the list I have placed the infections by mycelial organisms definitely different from the species *Actinomyces bovis* of Wolff-Israel. The condition known as "madura foot," or mycetoma, which we do not see in Europe, but which occurs fairly often in Asia and Africa and sometimes in America, is the most prominent member of this class. Here the infecting agent seems to be usually an aerobic species of actinomyces, often spoken of as *Actinomyces maduræ*, but there is reason to think that more than one type of organism can give rise to the disease. I am not aware that the source of these infections has been satisfactorily traced. Apart from madura foot a number of other cases have been reported from time to time in the literature, of both human and cattle infections by mycelial organisms quite distinct from *Actinomyces bovis*; for example, the case of urinary infection reported by Cohn [4], and the lung infection reported by Wynn [5]. I am very doubtful whether any true infections by *Actinomyces graminis*, the organism originally described by Bostroem [6], are on record. In Bostroem's own cases it is more than probable that he failed to cultivate the true infecting agent. I personally, have never met with a case of this kind.

The third group—a large one in the cattle diseases—is that of infections by the actinobacillus of Lignières and Spitz [7], a small Gram-negative organism, with characters totally different from those of the actinomyces. Apart from the fact that in certain media it takes on a filamentous habit of growth, just as many other bacilli do, there seems to be no reason for regarding it as a mycelial organism at all, but in the new American system of classification it is so regarded.

These infections have a particular interest because they comprise most of the so-called "woody tongues" of cattle, which for years have figured in our text-books as typical of true actinomycosis. After the first description of them by Lignières and Spitz in the Argentine little attention was paid to them until 1916

when F. Griffith [8], of the Local Government Board Laboratory, described his investigation of a series of tongues sent to him from Smithfield market. He found that by far the largest number of these were infected, not by actinomyces, but by the actinobacillus. Within the last few years his findings have been abundantly confirmed by Hülphers [9], Klarin [10] and Magnusson [11] in Sweden, Gunst [12] in Holland, and by T. J. Bosworth [13] in this country. So far as I know only one human case has been recorded (Ravaut and Pinoy, 1911 [14])—a case of meningitis—but it is not impossible that others have been overlooked.

About the fourth and fifth groups there will perhaps be more difference of opinion. It is claimed by the Swedish and Danish veterinary bacteriologists that there is a form of actinomycosis of the udder of the cow and the pig—in which granules and clubs occur but in which neither mycelial organisms nor the actinobacillus can be found. The granules are said to be composed of a dense mass of capsulated staphylococci and yield a pure culture of that organism which is indistinguishable from *Staphylococcus aureus* derived from other sources.

These infections in the cow and pig seem to be analogous to that known as botryomycosis in the horse—a granuloma also due to staphylococcus—arising about the spermatic cord after castration. I may mention one interesting point here with regard to the formation of clubs. Pus from the horse lesions (botryomycosis) shows discrete granules visible to the naked eye, but instead of a fringe of clubs at their periphery, they present a structureless hyaline zone. When, however, the staphylococci cultivated from these granules were inoculated into the testicles of guinea-pigs by Magrou [15] of the Pasteur Institute, they gave rise to suppuration and the pus showed granules with typical clubs. It seems probable, therefore, that infection by the staphylococcus can produce in one animal species (the cow or the pig or the guinea-pig) granules bearing clubs, and in another (the horse) granules without clubs.

Whether staphylococcus infections in man ever lead to club formation I cannot say, but it has been described in at least one instance, that of a French soldier with a war wound of the elbow.

In yet another class of case (group 5) met with in animal pathology, the granules yield only cultures of a diphtheroid bacillus which the veterinary bacteriologists have termed *B. pyogenes*. Magnusson [11] is disposed to regard this as another potential club-former, but he says that the evidence is not quite so strong as in the case of staphylococcus.

Lastly we have to consider the mycelial infections which are not productive of clubs (group 6 of Table I). They are by no means uncommon in human pathology but most of them go unrecorded.

Birt and Leishman [16] described a case of empyema in 1902, Henrici and Gardner [17] another lung case in 1921, and Blake [18] a case of septicæmia following a rat bite in 1918, and there are many other instances scattered through the literature. These cases form a heterogeneous group and often differ considerably from the ordinary *Actinomyces bovis* infections. I may briefly recall three cases from my own experience. The first was that of a man with a subacute abscess over the parotid gland from which foul pus was evacuated. It showed no naked-eye granules and gave a pure culture of an aerobic mycelial organism quite unlike actinomyces. The man recovered at once after the draining of the abscess.

The second case was that of a well-known scientific man who developed an empyema. The pus showed no naked-eye granules and gave a culture of staphylococcus and a mycelial organism. The latter quickly disappeared after the draining of the pleura and the patient made a complete recovery.

In the third case, that of a child, pus had been draining freely for several weeks from the peritoneum when I saw the patient, but she was not very ill. The illness had begun as an appendix abscess. Because a filamentous organism had been seen

in the pus, several consultants had diagnosed actinomycosis and given a bad prognosis. There was, however, none of the induration which is such a characteristic feature of true actinomycosis, nor were there any granules. The child ultimately made a good recovery. I should not have been surprised to see this case reported in the journals as an instance of remarkable recovery from actinomycosis and as proving the effectiveness of a particular remedy in that disease.

That is the end of my inventory. Doubtless it is incomplete, but it will serve to bring out two points which need to be emphasized.

The first point—and it is not at all new—is that the formation of clubs is not a specific character of mycelial infections. Thirty years ago it was shown that clubs were formed around a mass of dead tubercle bacilli implanted into living animal tissues, and since then this has been demonstrated in many other circumstances.

The second point is that the continued use of the term "actinomycosis" to cover all these different infections shown in Table I is unfortunate, and leads to confusion. If the meaning of words is to stand in any relation to their derivation, actinomycosis ought surely to connote an *infection by a filamentous organism which forms in the body definite bacterial colonies surrounded by a radial arrangement of clubs*. In this sense it is quite properly applied to groups 1 and 2 of Table I, but I think it should not be applied to group 3; and it is quite inapplicable to the infections of groups 4 and 5, which are not mycoses at all, although they produce clubs; equally it is inapplicable to group 6 which presents no clubs.

What is to be done to mend matters? Unfortunately, Magnusson [11] in his valuable paper on actinomycosis among animals, continues to use the term for all the ill-assorted group of infections. At the same time he reminds us of a suggestion which might be followed with advantage, namely, that of Lignières and Spitz [19] in 1903 that all this group of diverse conditions associated with clubs should be covered by the non-committal term "actinophytosis," and that where it was possible to specify the nature of the infecting agent, we should speak of "actinophytosis due to *Actinomyces bovis*, or actinobacillus, or staphylococcus," etc. That would at any rate make for precision.

Personally I should like to modify slightly the proposal of Lignières and Spitz in order to retain the established terms "actinomycosis," "actinobacillosis," etc., with strictly limited applications. And the term "paractinomycosis" (Colebrook, 1921) [20] might also be usefully retained to distinguish between the true actinomycotic infections due to *Actinomyces bovis* and those due to different species.

The several groups of infections shown on Table I would then be designated as shown on Table II.

TABLE II.—SUGGESTED NOMENCLATURE.

Actinophytosis (infections associated with granules bearing clubs) includes:
(1) Actinomycosis (infections by *A. bovis*).
(2) Paractinomycosis (infections by filamentous organisms other than *A. bovis*).
(3) Actinobacillosis (infections by the actinobacillus).
(4) Actinophytosis due to staphylococcus.
(5) Actinophytosis due to *B. pyogenes*.
Streptothricosis (infections by filamentous organisms but showing no granules, or granules with no clubs).
This group will have to be subdivided later—as bacteriological differentiation permits.

Before leaving this aspect of the subject I will refer to one curious result of the loose terminology that has grown up. We seem to have led the systematic botanists into error. There are many organisms in the soil which go by the name "Actinomyces "this" or "that." Apart from the fact that they are filamentous organisms, they have little in common with *Actinomyces bovis*, or any other mycelial organisms with which we are familiar in surgery. I have always been puzzled as to why the botanists called them "Actinomyces," since they show nothing analogous to the club formation we meet with in pathological conditions. The filaments will of

course, often stream out from a centre of growth, just as many other bacteria do, but that, I take it, is not what was originally meant by the prefix "actino." A few months ago I was told by a leading mycologist that the term "actinomyces" had been borrowed from pathology in the days when the association of these filamentous organisms with actinomyces had first been described.

On the strength of Bostroem's 200-page article [6] it had been assumed that actinomycosis was an infection derived from fungi present on grasses, cereals, etc., and when similar thread-forming organisms were discovered in the soil it seemed natural to designate them, at any rate provisionally, as "actinomyces." Now, although few people, apart from writers of textbooks, stand by Bostroem's hypothesis, and the mycelial organisms of the soil have never been proved to infect man or animals, the provisional name seems to be securely entrenched in systems of botanical classification.

The second question which might profitably be discussed is that of ætiology, to which I have just referred. I will briefly mention the important investigations recently made in Sweden by Dr. Naeslund.

For many years the presence of filamentous organisms in the human mouth has been recognized, e.g., on the teeth, in tartar, in carious cavities and in tonsillar crypts. Cultures of some of these thread-forms have occasionally been obtained, but until recently no one has seriously set to work to classify and investigate them. Indeed, the task seemed rather hopeless in view of the multitude of more freely growing bacterial forms always associated with the thread-forms. Dr. Naeslund [21] has, however, tackled the problem with some success, and by employing a medium containing sterilized saliva, has managed to isolate a number of mycelial types. Several of these he considers to be actinomyces, and some he is unable to distinguish by cultural and morphological characters from strains of *Actinomyces bovis* isolated from human actinomycosis.

The proof of identity of these strains is, of course, a matter of fundamental importance, and may be difficult to obtain. Even if they occasionally produce similar suppurative lesions in cattle, with "sulphur granules" and clubs, as Naeslund has reported, their identity is perhaps not beyond question. Probably a serological test will have to be the final court of appeal, and so far as I know, this has not yet been undertaken.

Dr. Naeslund has also been examining the relation of these thread-forming organisms to the formation of tartar [22] and salivary calculi. Sections of these concretions suggested to him that filaments of actinomyces and of leptothrix formed a sort of stroma in which the calcium salts were deposited in successive layers. This deposition is due, he thinks—and he brings experimental evidence in support of the view—to chemical and physical changes produced in the saliva in the immediate neighbourhood of the organisms, so that it can no longer hold all its calcium in solution. The most important of these changes is a shift of the pH towards the alkaline side. Naeslund believes further that the microbic filaments provide a suitable framework on which the precipitated salts can collect.

This short summary of work which has occupied several years is, of course, quite inadequate. If Dr. Naeslund's main conclusions are confirmed—as I think they probably will be—they will go far towards explaining the origin of true actinomycotic infections, as we see them in this country. Many cases are on record which point to the mouth as the probable source of these infections; in particular I may mention three: (1) the case reported by Israel [23] in which a fragment of carious tooth was found embedded in the middle of a focus of actinomycosis of the lung; (2) Mr. Cope's [24] case of actinomycosis of the knuckles which resulted directly from a blow that broke an antagonist's tooth; and (3) the case reported by Judd [25], which resulted from extraction of a tooth. The occasional presence of a vegetable

foreign body, such as a head of barley or of grass, in actinomycotic abscesses may seem at first sight to suggest that the infecting "fungus" has been introduced from without, but, upon reflection, it appears equally possible that in these cases the foreign body, impacted for a while in the mouth or pharynx, had become encrusted with a deposition of actinomyces previously present. Naeslund has demonstrated that such deposition upon foreign bodies does occur and may be the starting point of the formation of a salivary calculus [21].

In conclusion, I should like to express the hope that Dr. Naeslund's important investigations may be repeated and perhaps extended in this country.

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Mr. Zachary Cope: The common form of actinomycosis in man is that caused by (or at least associated with) a Gram-positive anaerobic hyphomycete, which at some stage of its growth in the tissues, forms small granules which are to be seen in any softened focus, and which are composed of a feltwork of the fungus usually surrounded by a radiating series of Gram-negative club-like processes. The recent work of Bosworth has shown that a considerable proportion (13 out of 34) of the cases of actinomycosis in cattle are caused by a similar organism, and it is to the effects of this anaerobic, Gram-positive hyphomycete that we must limit our remarks, inasmuch as the commoner actinobacillus of cattle has hardly ever been known to affect man.

Actinomycosis is to my mind the most interesting of the granulomata, and its study is full of puzzling problems. The advance of our knowledge of the subject during the last fifty years has been negligible, compared with the wonderful steps forward in the case of syphilis and tuberculosis.

In these remarks I wish rather to emphasize the strange features of the disease which call for investigation rather than to recapitulate facts which you already know.

The first problem of interest to the surgeon is that of the origin of the fungus. What is its habitat? How does it reach the tissues? The work of Lord and of Homer Wright carries conviction that it is a common, if not usual, inhabitant of the mouth and resides in many carious teeth. There is no difficulty in understanding how slight abrasions of the cheek and gums will enable the fungus to enter the tissues, nor is it any cause for surprise that the cæcum and appendix should contain swallowed portions of the mycelium. Similarly, minute aspirated fragments might conceivably account for the pulmonary lesions, or alternatively, the fungus might creep through small lesions of the lower œsophagus and infect the mediastinal tissues. The problem of how the fungus gains access to the mouth and teeth still, however, remains. The current view that infection comes from cereals and grasses is difficult to maintain, since no anaerobic fungus of a similar nature has been isolated from this source. In spite of this fact there are many clinical cases in which a direct connection may be traced between barley and other grains and actinomycosis, and further investigation is needed on the point.

The age incidence of the disease must surely have some significance. It is very rare for a child under five years to be attacked. Can it be that there is any relation between the earliest incidence of the disease and the onset of dental caries? It may well be.

The geographical distribution of actinomycosis calls for little comment except in one respect. By the courtesy of the Registrars of hospitals in different parts of England and Scotland, I have obtained figures showing the number of cases of the disease admitted to the wards of these hospitals during a period of years. These show that in most parts about one case in 3,000 total in-patients is suffering from actinomycosis (about one in 1,600 of surgical admissions). But in Edinburgh and Glasgow the admissions for actinomycosis are only about one quarter what they should be by this ratio of admissions, and one is left to wonder whether this is a merely accidental or a significant variation.

NUMBER OF ADMISSIONS FOR ACTINOMYCOSIS AT VARIOUS HOSPITALS.

	Cases	In years
St. Bartholomew's Hospital	27 ...	10
Middlesex Hospital	25 ...	10
Glasgow Royal Infirmary	5 ...	10
Birmingham General Hospital	20 ...	10
University College Hospital	9 ...	9
Edinburgh Royal Infirmary	8 ...	9
Guy's Hospital	15 ...	7
Newcastle-on-Tyne Infirmary	14 ...	7
Charing Cross Hospital	6 ...	4
King's College Hospital	14 ...	11
St. Thomas's Hospital	15 ...	10
St. Mary's Hospital	15 ...	10
Liverpool Royal Infirmary	9 ...	10

From the figures for in-patients from the various hospitals one gets rather a wrong idea of the sites of incidence of the disease. Of the 182 cases mentioned in the statistics quoted above, we find that 80 involved the face and neck, 28, the chest, 62, the abdomen, and 12, various other parts. But the majority of cervico-facial cases do not need admission to hospital, and in complete figures, comprising out-patients, this group of cases would be much larger.

Concerning the conduct of the actinomycotic fungus in the tissues of the body, there are three points which are peculiar and characteristic. The first is the way in which the process advances almost always by contiguity of tissue. Blood transmission occasionally occurs, as for example, infection of the liver from the appendix through the portal veins, but usually the slow inflammation progresses by contiguity of tissue, often receding in one place and advancing in another. The second characteristic is the curious immunity of the lymph-glands to the infection. I have never seen a lymphatic gland infected by actinomycosis, and have not seen any authentic record of such a happening. This feature places it in sharp contrast, not only with the actinobacillosis infection of cattle, but also with almost every other variety of infection both acute and chronic. Thirdly, everyone who has seen much of actinomycosis must have pondered over the remarkable reaction which the fungus causes in the connective tissues. I think it was Unna who pointed out that here we have an almost unique example of tissue reaction at a distance, for the changes may take place at a considerable distance from the place where the fungus is situated, and it is possible to cut many sections of the curiously hard, gristly, fibrous tissue without finding any filaments of the parasite. This connective tissue reaction differs considerably from that found in tuberculosis and syphilis, not only in the relative proportion of the various kinds of cells found in it, but also in the fact that the blood-vessels are not obliterated. Exactly how the fungus causes this reaction is unknown. That it is a beneficial reaction is very likely, for it is noteworthy that in those parts where connective tissue is comparatively scanty in proportion to the

epithelial elements of the organ, e.g., in the lungs and liver, the prognosis is much more grave.

It is unnecessary here to do more than outline the clinical picture of actinomycosis as it affects the various parts of the body. When it affects the face and neck the picture is usually that of an infiltrating inflammatory mass in the parotid region, around the lower or upper jaw, or in the submaxillary or lower cervical regions. Occasionally it spreads to the deeper tissues and may enter the skull and erode the vertebræ. Trismus is common, but pain, as a rule, is slight. The lower jaw may be eroded externally or, occasionally, may be primarily involved. In every case, softening of the inflammatory mass ultimately occurs and granules can then be obtained from the pus. Softening is sometimes delayed for several months during which time diagnosis is difficult.

In the right iliac fossa, infection almost always spreads from a diseased appendix, usually after the removal of a perforated appendix, but occasionally when no operation has been performed. A persistent sinus with indurated borders, or a large, hard, and rather fixed mass may indicate the condition. The disease does not readily invade the peritoneum but spreads retroperitoneally. The liver may be affected secondarily by portal metastasis and the symptoms of this affection may not show for many months, or even for a year or two, after the attack of appendicitis.

Thoracic actinomycosis may take the form of a chronic bronchitis but more commonly the inflammatory infiltration appears to spread from the mediastinal tissues to the pleura and lung, and generally comes to the surface in the form of a subcutaneous abscess from which the granules can be obtained. The base of the lung is more commonly affected than the apex, and the initial surface-abscess is usually about the level of the diaphragmatic attachment to the ribs.

The brain, kidney and other viscera are sometimes the seat of actinomycosis: infection must generally be by the blood-stream in these cases. The ovary may be affected by contiguity across the peritoneal cavity.

Primary actinomycosis of the skin and subcutaneous tissues is not common, but sometimes results from penetrating wounds. It may take the form of an indolent inflammation discharging through one or more sinuses; occasionally a tumour formation composed of granulation tissue may project from the skin surface and present several discharging sinuses from which the fungus may be obtained. The skin is often involved secondarily to the underlying tissues.

Diagnosis of actinomycosis is only made with certainty by the finding of granules of the fungus in the pus or by seeing them in sections of the tissue. It is not sufficient to find granules—they must be examined microscopically and show the typical Gram-positive mycelium and, preferably, clubs also. Cell débris can sometimes closely simulate the naked-eye appearance of a fungoid granule.

Granules, however, are only found when the inflammatory tissue has softened, so that there is often a time—occasionally a long time—when diagnosis has to be made provisionally, on clinical grounds. It is often possible to diagnose the condition with a reasonable degree of certainty long before the fungus can be found. Distinction has to be made from chronic sepsis, tubercle, syphilis and new growth. Tubercle tends to soften and ulcerate earlier than actinomycosis. Syphilis will give a positive Wassermann reaction. Sepsis is more acute, may cause sequestra of bone and soon comes to an abscess. It may be impossible to exclude new growth unless a section of the tissue is examined.

The combination of actinomycosis either with sepsis or with malignant disease may make the diagnosis more difficult. Septic organisms tend to spread the inflammation, but diminish the activities of the ray-fungus and render its detection less easy. In a case recorded by Wakeley malignant disease was superadded to actinomycosis.

The difficulty which may be experienced in diagnosis is well illustrated by the case of Tilanus, which is quoted in Poncet's book. It was that of a young woman who had on the left cheek a lesion which was thought at first to be a syphilitic gumma. As it advanced in spite of several weeks' treatment with potassium iodide, the diagnosis was altered to that of malignant disease. Then, because of the slow progress and slight tendency to invade the surrounding parts, the conclusion had just been reached that the condition was tuberculous, when the true nature of the disease was revealed by the discovery of the actinomycotic fungus in scrapings from the sinus.

The only safe rule is to consider the possibility of actinomycosis in the diagnosis of every chronic inflammatory or supposed neoplastic swelling, particularly in the region of the face, jaws and right iliac region. In the chest the disease is unlikely to be diagnosed before the fungus is found either in the sputum or in the pus from opened abscesses, for the early stages of thoracic actinomycosis are very insidious.

The prognosis of actinomycosis varies greatly according to the part of the body affected. The most favourable site is the cervico-facial region, for here the majority of the lesions become healed in course of time. Only comparatively rarely does the fungus metastasize to a vital part, or track upwards to the skull or downwards to the mediastinum and so become perilous.

Less favourable is the outlook in ileo-cæcal actinomycosis, but even here about half the cases appear to recover. If the fungus gains access to the liver via the portal radicles, the prognosis is almost hopeless.

Thoracic actinomycosis sometimes takes the form of a bronchitis without any solidification of the lung; in these cases recovery is quite possible and likely. Sometimes also the subpleural tissues are infected and a local abscess in the region of one of the ribs may develop; here also one would expect recovery.

But when the parenchyma of the lung is involved, and the mediastinal tissues are infiltrated, the ultimate outlook is exceedingly grave, though life may be prolonged for a year or two.

Treatment.—Up till now there has been discovered no certain specific for actinomycosis comparable to salvarsan in the treatment of syphilis. Most cases are treated by a combination of methods which makes it difficult to appraise the merits of each method individually.

First we must mention the administration of iodine in one form or another. Potassium iodide is the best known drug and this may be given in doses up to as much as a hundred grains a day. Though I admire the faith and courage of those who advance the dose up to 800 or 1,000 gr. a day, I would not follow their example unless more definite evidence of its efficacy were forthcoming. A palatable way of giving iodine is to administer tincture of iodine in milk, whilst the collosol form of the drug is very convenient. Iodipin and tiodine are two other compounds which have been used with effect. Good results often follow, but how much of the cure is due to the drug is difficult to determine.

Intramuscular and intravenous injections of colloidal copper have been tried with apparent benefit, but here again it is difficult to obtain controlled evidence. Salvarsan has been tried with doubtful results.

Clinical evidence supports the view that X-rays have a softening influence on the hard mass of inflammatory tissue so often found in the disease. Radium is less certain and less easy to apply evenly throughout a lesion.

Specific therapy by means of vaccines has been tried but the results have not been striking enough to convince one of their efficacy.

Surgery has a definite sphere in the opening of abscesses as they develop, in the removing of sequestra as they may form (in the jaw) and in the removal of some of the dense gristly mass of connective tissue which sometimes remains for months as an indolent tumour showing little tendency to soften.

The injection of a solution of formalin into the swelling has been recommended in cases involving the face and neck. This is a painful process necessitating an anæsthetic, and I see no advantage in recommending this method when other lines of treatment are open.

Though actinomycosis sometimes forms large retroperitoneal masses which project into the peritoneal cavity, we have never yet heard or read of any obstruction of the intestine caused thereby, so that the need for intestinal anastomosis must seldom, if ever, arise.

Mr. T. J. Bosworth (*Institute of Animal Pathology, Cambridge*): Actinomycosis is a condition of considerable economic importance in certain species of animals. The term is ordinarily used in a wide sense, to include a number of distinct pathological entities which in their clinical aspect resemble each other closely and can only be distinguished with certainty by microscopic and cultural examination. It is thus applied to all progressive infections of a chronic character showing lesions of the suppurative type, associated with the formation of granulation tissue in which the causal organism appears in the form of colonies or granules possessed of the so-called clubs. This practice is obviously open to objection but it is easy to see how it has arisen. The existence of club-bearing granules was first noted in cases of true actinomycosis both in man and animals and it appears to have been assumed for quite a number of years that the streptothrix organism responsible for that disease was the only one capable of producing such colonies. Thus the mere presence of club-shaped elements came to be regarded as diagnostic of actinomycosis, and when it was shown that this view was erroneous, the term "actinomycosis" was applied in a more general sense to include all such cases. As long ago as 1902, Lignières and Spitz showed that a disease of cattle in the Argentine which would ordinarily have been regarded as typical actinomycosis, on account of the character of the lesions and the presence of club-bearing granules, was due to a small Gram-negative bacillus to which they gave the name "actinobacillus." Since that time, as the result of numerous investigations, it has been established that this organism is the cause of a large proportion of cases of the disease in many parts of the world.

Then again Magrou has shown experimentally that staphylococci are capable of producing lesions containing club-bearing colonies and it is now known that chronic staphylococcic infections of this type occur naturally in the mammary glands of the cow and the sow, and have in the past been regarded by many as being of an actinomycotic nature.

Thus there are at least three distinct types of infection in animals, characterized by the presence of club-bearing granules to which the term "actinomycosis" is somewhat loosely applied. It ought to be emphasized, however, that current teaching on the subject—in this country at any rate—lays due stress on the necessity of differentiating such conditions according to their ætiology and regarding each as a separate and distinct disease. It also recognizes the fact that in many cases certain clinical features afford a fairly reliable guide as to the particular infection responsible. Thus, lesions of the tongue, pharynx and glands of the head in cattle are almost invariably due to the actinobacillus, whilst *Actinomyces bovis* is to be suspected in cases when the jaw bones are affected and the regional lymphatic glands are not involved. Much of the confusion that previously existed has therefore disappeared, though it is unfortunate that the descriptions given in the more important textbooks are still based on the old idea of the paramount importance of *Actinomyces bovis*, with the result that among the lesions caused by this parasite are included many that are due to other organisms. The subject of actinobacillosis is accordingly dismissed in a few paragraphs which shows that its relative importance has been insufficiently realized by the authors, whilst no reference whatever is made to chronic staphylococcic infections in which actiniform colonies are present.

It is very desirable that the facts with regard to the occurrence of these various types of infection should receive general recognition and that each should be regarded as a separate disease and named accordingly.

The use of the term "actinomycosis" should, as suggested by Wright, "be restricted to a suppurative process combined with granulation tissue formation, the pus of which contains the characteristic granules composed of dense aggregates of branched filamentous micro-organisms and of their transformation or degeneration products." Such products may be present in the form of club-shaped elements or may show no definite morphology. The condition as thus defined, is to be distinguished from actinobacillosis and also from chronic lesions, due to staphylococci, in which granules are present. In view of the work of Magrou it appears justifiable to include all such staphylococcic cases under the term "botryomycosis," irrespective of the presence or absence of club formation.

An alternative form of nomenclature was put forward in 1903 by Lignières and Spitz, who proposed the word "actinophytosis" as a generic name for all infections in which club-bearing granules are present, and suggested that each of its various forms should be denoted by reference to the organism responsible, e.g., actinophytosis due to actinobacillus. In my opinion the use of such a generic term is to be avoided, as it is likely to convey the impression that there is some essential relationship between the various conditions to which it is applied.

I will now give a brief survey of the situation so far as the various domesticated animals are concerned.

Cattle.—The bovine species is affected much more frequently than any other, and is subject to each of the three diseases under discussion. As the result of a number of investigations which have been conducted since the publication of the original work of Lignières and Spitz, it has been fully established that cases of actinobacillosis are, in general, more common than those of actinomycosis, whilst botryomycosis is a comparatively rare condition usually confined to the udder.

(A) Actinobacillosis: The lesions of this disease are to be found affecting the head and upper part of the neck in a very high percentage of cases, and are often confined to this region. They may occur, however, in connection with the skin in various parts of the body and also in the internal organs, among which may be mentioned the wall of the stomachs and intestines, the lungs, liver and serous membranes. The actinobacillus, in contrast to the *Actinomyces bovis*, shows a tendency to invade the soft tissues and to spread by way of the lymphatics. It is rarely found in bone lesions. Thus in the head the primary lesions occur in the tongue, gums, hard palate, the wall of the pharynx, the cheeks, lips and muzzle. Infection quickly spreads to the lymphatic glands, which tend to become enlarged and prominent. In advanced cases they are converted into fluctuating abscesses, filled with thick, glutinous greenish-yellow pus, in which the characteristic granules are very abundant. It is possible to make a reasonably accurate diagnosis by simple microscopic examination of the pus. Having ascertained that typical club-bearing granules are present in a fresh preparation, it only remains to crush a small quantity between two slides and apply a Gram stain. The absence of Gram-positive elements is strongly suggestive of actinobacillosis, though the actual organism itself cannot always be distinguished with certainty as it is largely obscured by the amount of débris present in such a preparation. It can, however, be recovered quite easily in culture by sowing crushed pus on to agar tubes and incubating aerobically at body temperature. A good growth is obtained in about eighteen hours, and this is usually found to be quite pure, provided fresh material was used for inoculation. The virulence of freshly isolated strains varies considerably. Lignières and Spitz studied the development of the organism in experimentally produced subcutaneous lesions in cattle, and also showed that purulent nodules containing masses of typical granules

resulted from intraperitoneal injection of culture into the guinea-pig. Magnusson, in a recent interesting publication on actinomycosis in general, records that of thirty-seven strains inoculated into cattle, ten gave rise to definite lesions of actinobacillosis and the rest gave negative results, whilst six were inoculated into guinea-pigs (five animals being used for each strain) without producing any effect. Of my own strains only one proved capable of producing effects in guinea-pigs comparable to those described by Lignières and Spitz. Actinobacillosis may be diagnosed in the living animal by the agglutination test. This is sometimes of value in practice, as a means of distinguishing the condition from tuberculosis when considering the desirability of initiating treatment. According to Magnusson, affected animals show a titre of from 1 : 60 to 1 : 320 or more, whilst normal serum does not agglutinate in 1 : 20.

(B) Actinomycosis : The common form of this disease in cattle begins as an infection of the jawbone, and the available evidence strongly suggests that primary actinomycosis in other situations is extremely rare. This is certainly true so far as the head is concerned, for this is the region which has provided most of the material used by investigators. It is probably to a large extent true also as regards other parts of the body, although precise information on this point is rather scanty. However, if we consider, as a whole, the results obtained within recent years by three independent investigators—Hülphers, Gunst and Magnusson—we find that out of 396 so-called actinomycotic lesions examined, 31 were obtained from situations such as the lung, udder, stomach, peritoneum and skin, not one of which proved to be true actinomycosis. That the disease does occasionally occur in such situations must, however, be admitted, though recorded cases are few. In the case of the jawbone, infection probably gains entrance through the alveoli in many instances, or it may reach the periosteum through an ulcer of the gums. It sets up a chronic suppurative inflammation resulting in destruction and rarefaction of bone and its replacement by granulation tissue containing purulent foci. At the margin of the lesion new bone is produced, and the combined processes ultimately lead to considerable enlargement of the jaw. The active process may extend to the overlying soft tissues, and ultimately reach the skin or mucous membrane causing perforation and forming a fistula which discharges pus. The pus is much thinner in consistency than that of actinobacillosis, and the granules it contains are usually larger, and show a much greater tendency to become calcified. *Actinomyces bovis* shows no tendency to spread by way of the lymphatics and so invade the lymph glands. In cases of some standing, however, the glands are to some extent enlarged and of increased consistency, as the result of a chronic lymphadenitis which is probably a reaction to toxic substances absorbed from the lesions.

The causal organism of actinomycosis was first isolated and described by Wolff and Israel in 1891. In smears from crushed granules it appears as a mixture of Gram-positive elements in the form of branching filaments, bacillary and coccoid forms. It grows only at body temperature under conditions of partial anaerobiosis. The colonies first appear in surface cultures after two or three days' incubation, in the form of tiny dewdrops. On further incubation they became opaque, whitish or yellowish-white, and rather granular in appearance, and increase in size up to one or two millimetres in diameter. They are easily picked off the medium. In smears from cultures the organism is pleomorphic, and appears chiefly as an ovoid or rod-shaped Gram-positive bacterium showing somewhat uneven staining. Filamentous forms are sometimes observed, but branching elements are only rarely present. This organism is thus sharply differentiated from that described by Bostroem as the cause of actinomycosis, which is now considered to have been a saprophytic contaminant of the lesions.

Personal experience would suggest that actinomycotic lesions, especially when perforation of a free surface has occurred, are apt to become contaminated by various

bacteria, principally staphylococci, streptococci and *B. pyogenes*, the presence of which may render difficult the isolation of the causal organism. On the other hand, the cases examined by Magnusson were remarkably free from such contamination, and he was able to obtain a pure culture from 54 out of a total of 61. The organism described by Colebrook under the name of *Actinomyces comitans*, which is commonly present in association with the granules of actinomycosis in man, has not been observed, so far as I am aware, in bovine lesions.

There have been a number of attempts by various workers to reproduce actinomycosis experimentally. Most of these have failed entirely, or have resulted in the production of relatively insignificant lesions showing little tendency to progress.

Better success has attended the efforts of Magnusson as the result of a fairly large series of inoculations. Experiments on cattle with material from thirty-two cases, yielded positive results in eight instances. It was found that an interval of several months may elapse after inoculation of a pure culture before a typical actinomycotic growth is obtained. The surgical removal of a portion of the growth seemed to hasten the development of the process.

(c) Botryomycosis.—This condition is occasionally observed in the cow's udder and may be mistaken for tuberculosis of that organ. It was formerly regarded as actinomycosis owing to the presence of granules with well-defined clubs. It has been shown, however, that the granules consist essentially of masses of staphylococci, and the condition is therefore botryomycosis.

Swine.—In these animals the mammary glands are most commonly involved and lesions elsewhere are comparatively rare. Actinomycosis of the lungs has, however, been reported in Australia. Lesions have also been observed in other parts of the body but there is insufficient evidence upon which to base an opinion as to their real nature.

In the article by Magnusson to which reference has already been made, the results of the examination of a large number of udder lesions in swine are recorded. The condition appears to be very prevalent in Sweden, for according to the author's estimate, about 25 per cent. of the older sows were found to be affected in the slaughter-house at Malmo. The results showed that 181 of these cases were actinomycosis whilst 41 were botryomycosis. An examination of the strains isolated from the former showed that it was possible to divide them into two groups on account of slight differences in their cultural characters, and on similar grounds they could be separated from cattle strains. It is therefore necessary to recognize at least three types of *Streptothrix israeli*.

In cases of botryomycosis the granules were very varied in size and shape. Some were provided with a complete ring of clubs, some were quite devoid of these structures, whilst others possessed them only in parts.

Horses.—Actinomycosis is only occasionally seen in horses, the commonest sites of the lesions being the head and the spermatic cord. Further investigation of the characters of the causal organism in this species is desirable, as it is by no means certain that *Streptothrix israeli* is the ætiological factor. My own experience extends to two cases only and here the organism was found in the lesions in the form of typical granules which, however, were devoid of clubs. In culture both strains were aerobic and grew only at body temperature. Surface-growths occurred in the form of dry, heaped-up, wrinkled colonies which were adherent to the medium and attained a maximum diameter of 3 or 4 millimetres. In one case the colonies were pure white, whilst in the other they assumed a dirty yellow colour. In smears made from cultures these organisms appeared as Gram-positive branching filaments. In order to keep the strains alive it was necessary to subcultivate every two or three weeks.

Botryomycosis is a very well known condition in the horse, in which animal it was first described. The typical granules present in the pus are devoid of clubs and

consist of masses of staphylococci surrounded by a narrow zone of acidophile material of homogeneous appearance. These organisms were at one time thought to have special characters not possessed by ordinary staphylococci, but this view has been shown to be erroneous by Magrou whose work has been of great value in elucidating the ætiology of the condition.

Other Species.—Lesions of the type under discussion are absolutely rare in other species of animals, but actinomycosis, or at least streptothricosis, has been reported in dogs and cats, and actinobacillosis in sheep.

Dr. F. Parkes Weber referred to the connection between actinomycosis and sialolithiasis. He was at present interested in a man (J. C.), aged 36, who attended the out-patient department of the German Hospital for chronic indurated inflammation on the left side of the neck (cervico-facial), with ulceration and the formation of a purulent sinus, without any obvious involvement of lymphatic glands. The appearance had suggested actinomycosis, but microscopical examination of granulation tissue from the sinus showed no ray-fungus, but a great number of very large "macrophages" whose cytoplasm was full of polymorphonuclear leucocytes—an appearance similar to that of blastomycotic tissue, as figured by A. Buschke in Kolle and Wassermann's *Handbuch der path. Mikroorganismen*, second edition, 1913, v, pp. 172 and 173, figures 5 and 6. Definite improvement followed treatment with potassium iodide. On December 20, 1929, a small cherry-sized spherical calculus was excised by Mr. H. Rast from the left sublingual salivary gland, and the potassium iodide was temporarily discontinued. The inflammatory trouble on the left side of the neck became worse whilst the potassium iodide was omitted, but no certain microscopic nor cultural evidence of either actinomycosis or blastomycosis had been obtained (up to January 20). The patient was otherwise healthy, without any signs of tuberculosis and with negative Wassermann and Meinicke reactions.

If G. Söderlund (*Acta Chirurgica Scand.*, 1927, lxiii, supp. ix, pp. 1-237) was right or only half right, in regard to his belief that practically all salivary calculi were the result of a "ductogenic" actinomyces infection, a large class of benign cases would have to be added to the well-known relatively grave cases, as being due to infection with some kind of actinomyces. In these benign (sialolithic) cases the actinomyces was apparently seldom able to attack the tissues outside the salivary ducts. If some kind of actinomyces were really a frequent cause of salivary calculi, the fungus growing within the ducts would have to be considered as in some way collecting the calcium from the saliva and itself acting as a foreign body around which the calculus could be deposited in concentric layers. The present case, though not yet proved to be mycotic, suggested that a relatively harmless growth of fungus within the salivary ducts might occasionally be associated with an involvement of the superficial soft parts of the side of the neck and face.

Mr. Philip Turner said that actinomycosis was a subject of great interest to every general surgeon. The disease spread in a characteristic way, healing in one part and breaking out elsewhere. A very striking feature of the spread was the way in which the disease leapt over what were usually regarded as very definite anatomical boundaries. For instance he had recently had a case of actinomycosis of the liver in which the disease had directly extended through the diaphragm to invade the pleural cavities and the lungs. Cases were encountered occasionally in which organs such as the testicle or kidney, or even the central nervous system, were involved, and sometimes no other lesion could be found. In these cases it must be assumed that there had been some primary deposit which possibly had disappeared altogether, and that the lesion found was either due to a metastatic deposit or else to direct extension with complete disappearance of the original focus. He always made it a rule, in a case of obscure suppuration in the abdominal cavity, to bear in mind the possibility of actinomycosis as the cause.

Dr. Mervyn Gordon said it would be a great gain if serology could be applied to the diagnosis of actinomycotic infection. He had examined a great deal of material from cases in which the nature of the infection was doubtful; and repeatedly he had found actinomyces where its presence had been unsuspected. He thought that this must be the experience of others who made many bacteriological examinations in a large hospital. One private case that had impressed him particularly had been seen by about thirty consultants, and it was only shortly before death that pus obtained from a discharging sinus was found to contain the actinomyces organism in pure culture. In another case in which the patient was thought to be suffering from tuberculosis of the spine, pus from a discharging abscess was examined because material was required for a bacteriological class. In that case the diagnosis of actinomycosis was confirmed independently by another observer who made the post-mortem examination.

Often there was a real difficulty in obtaining granules; and under those conditions the only safe course was to regard the case, temporarily at least, as one of actinomycotic infection. In most of such cases, in which sections were afterwards forthcoming, this preliminary diagnosis was microscopically confirmed.

It was important to identify the cases of infection by the "No. 1" streptothrix, and in this he was sure serology could help. It would make for progress if cultures or material from cases of actinomycotic infection could be sent to some centre. There would then be an opportunity to see how many different kinds and types were at work, and thus advances could be made.

Mr. E. W. Riches' notes of two cases brought forward to show two modes of successful treatment are as follows:—

(I) In this case improvement only followed the use of a vaccine. The patient was a woman aged 27, admitted to Middlesex Hospital under Mr. Vaughan Hudson. A month previously she had had the last lower molar tooth on the left side extracted, but the root had been left

FIG. 1.—*Case I.* Granuloma at root of last molar. (Mr. E. W. Riches.)

behind; the adjacent second molar had been removed a week before. There was a tender swelling over the left mandible, with trismus, and the signs were those of a chronic aveolar abscess. X-rays showed the broken-off root and a small clear area around it, which was diagnosed by Dr. F. G. Nicholas as a root abscess or a granuloma (fig. 1). The root was extracted under a general anæsthetic, but no pus was found. Two days later the swelling in the left side of the neck was incised and drained and an ounce of pus removed; no streptothrix was found. A few days later the swelling was again incised and the pus re-examined, and Dr. L. E. H. Whitby reported as follows: "Specimen contained granules which consisted of a branched mycelium, which showed clubbing and resembled streptothrix actinomyces."

The patient had already been having potassium iodide and the dose was increased up to 150 gr. a day. After further incisions had been made—one inside the mouth—she showed

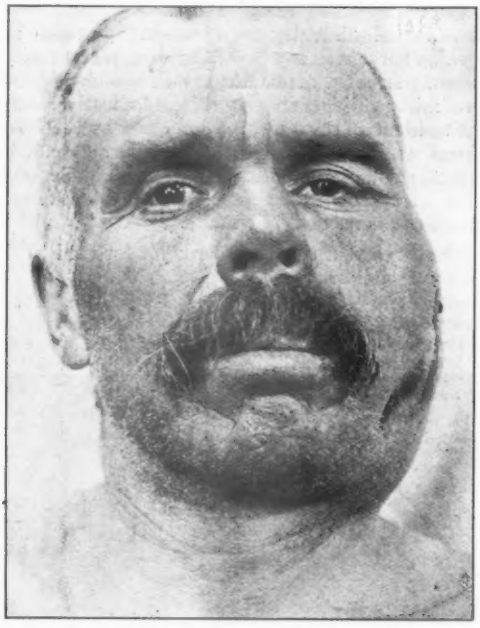

FIG. 2.

Case II.—Before treatment. (Mr. E. W. Riches.)

some signs of improvement and was discharged to a convalescent home after six weeks in hospital. A fortnight later she was sent back; there was now a brawny swelling covering the whole of the left side of the neck, with two sinuses and an area of fluctuation. Three further incisions were made and a stock actinomycosis vaccine was given; the initial dose was one million (mycelial threads) and this was increased at bi-weekly and later tri-weekly intervals up to one hundred millions. After a few doses she began to show definite improvement, and after seventeen doses in all, four of which were of one hundred millions, she was discharged healed. When seen three months later she was clinically cured; there was no swelling or sinus and the scars were scarcely visible.

(II) The other case had been successfully treated by means of buried radium. The patient was a farm labourer aged 52, admitted to Middlesex Hospital under Mr. Sampson Handley in 1928, complaining of a swelling on the left side of the face, with a discharging

sinus. Twenty years before, he had had a dental abscess which had discharged intermittently ever since. Two months before he came up the swelling increased in size, became painful and burst after six weeks, and a further swelling had recently appeared and was behaving in the same way.

On admission, there was an irregular swelling on the left side of the face, extending from the zygomatic arch to below the angle of the jaw, and from the angle of the mouth to the mastoid process (fig. 2). Two inches behind the angle of the mouth was a sinus discharging pus, and further back was a circular ulcer one half an inch in diameter, its base covered with

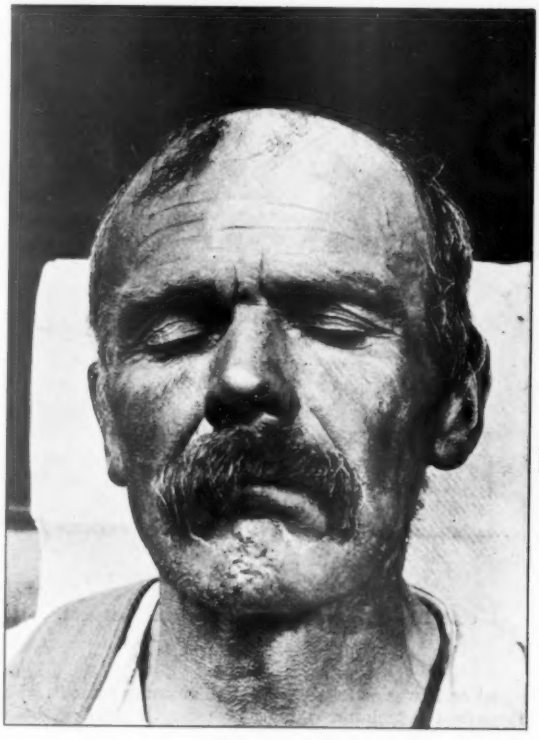

FIG. 3.

Case II.—Thirteen months after radium treatment.

purulent granulations. In front of the ear was a red prominent fluctuating area, whilst the remainder of the tumour was firm and nodular. The seventh and eighth cranial nerves were intact.

Examination of the pus showed no organisms in film or culture, and a provisional diagnosis of (?) neoplasm of parotid; (?) actinomycosis was made. After examining a second specimen Dr. Whitby reported that it contained a small quantity of unbranched mycelium, but that a more copious specimen would be required to establish a diagnosis of actinomycosis.

Radium tubes were then buried in the tumour, a dose of 8,400 mgm. hours encircling the mass, and a week later a further dose of 1,200 mgm. hours into its centre. More pus was

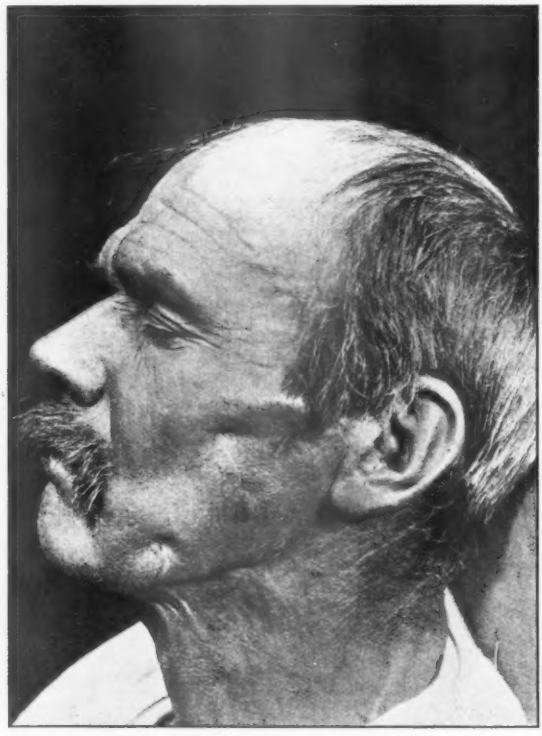

FIG. 4.

Case II.—Thirteen months after radium treatment.

obtained, and the report on it was as follows: "Small granules present. Films contain a fair quantity of an unbranched mycelium, which, save for the lack of branching, resembled streptothrix actinomyces. No other organisms in films or cultures." He was discharged after three weeks in hospital, taking potassium iodide to the amount of 75 gr. daily. Thirteen months later he reported, and was clinically cured; there was no swelling and the sinuses were healed (figs. 3 and 4).

Although the bacteriological diagnosis in this case was not absolute, the chronicity and clinical features made a diagnosis of actinomycosis fairly certain; the case is brought forward to suggest that radium might be tried in cases which proved intractable to other measures.

Mr. G. Dunlop Martin said that years ago, when he was engaged in country veterinary practice, there were in his district many cases of actinomycosis in cattle; and he recalled that while considerable improvement resulted from large doses of potassium iodide, he found a much more rapid benefit from adding rather heavy doses of biniodide of mercury.

Sir Holburt Waring said that his experience in connection with true actinomycosis had been mainly with regard to the abdomen and the face and neck and had shown that in the abdominal region by far the most usual site was the right iliac fossa and also that the appendix was not the primary seat of the infection. Some years ago he had encountered a number of these cases, and examined them carefully; and the conclusion at which he had then arrived was that the primary disease was in the wall of the cæcum, in the region of the appendix. This fact might afford an explanation of some cases in which, when the appendix was removed, no sign of actinomycosis could be found.

With reference to diagnosis: The hardness of these swellings, on palpation, was such that, to those who had seen a number of these cases in the early stages, there was not much liability to mistake.

Several years ago a patient had been sent to him by a medical colleague on account of a swelling in the right iliac fossa. The doctor had diagnosed carcinoma of the cæcum, and he (Sir Holburt) had agreed with the diagnosis. At operation, when he had exposed the cæcum, he had found a swelling having the naked-eye appearance of malignant disease, and when he placed his finger on it, the characteristic hardness associated with actinomycosis was evident. It was not feasible to excise the mass, because of dense adhesions to surrounding structures, but he removed a small piece for examination; the condition proved to be actinomycosis. The patient was treated with large doses of potassium iodide. One or two speakers had referred to 100 and 150 gr. of iodide of potassium being large doses—one even referred to 800 gr.; in this case the dose had been increased up to 480 gr. per day, and under that administration the swelling entirely disappeared. The patient passed out of the hospital's ken. Twenty years later he (Sir Holburt) was told that this patient had gone into the hospital again and had died there, the cause of death having been carcinoma of the stomach. He had examined the cæcum and vermiform process, which were removed at autopsy, and he did not find any sign of disease. The specimen had been in St. Bartholomew's Hospital Museum, and might still be there. It illustrated a result of the drug treatment of the disease.

He had not found that vaccines were of any definite advantage in treating this disease. The only satisfactory treatment he had seen was excision, when that was possible, and evacuation of abscesses and scraping, accompanied by the administration of potassium iodide. He had never used a combination of mercury with potassium iodide. He had not observed any beneficial results from treatment by X-rays.

One curious point which he had noted in connection with neck lesions was that if the actinomycotic focus was the site of a secondary infection, it was difficult to find actinomyces, but if some portion of tissue could be found which had not yet become infected with staphylococci or streptococci, actinomyces could often be found in it. He had always tried to secure a portion of tissue which had not been secondarily invaded.

Another point concerned the making of a preliminary diagnosis of actinomycosis when the abscess was opened. As had been stated, the contents of the abscess cavity were glutinous, and when the abscess was in the liver there was almost always a jelly-like tenacious material, which was more or less characteristic of the disease.

He had not been able to understand an occurrence in one of his cases, in which there was actinomycosis of the cæcum and an abscess in the iliac fossa. In the affected tissues there were typical yellow granules, which were found, bacteriologically, to be actinomyces. Some time afterwards the patient had died. At post-mortem examination, abscesses were found in the liver having the definite characteristics, but the granules were coal black. He had seen such black granules twice, and did not know the explanation.

He had never yet seen a patient recover from actinomycosis of the chest or the liver. He had seen a considerable number of recoveries when the disease was in the neck, face, etc., and a fair number when it was in the right iliac fossa.

He had had an extensive experience of salivary calculi of the parotid and the submaxillary duct and salivary glands, but he had never seen salivary calculi associated with actinomycosis.

Dr. Parkes Weber suggested that the extraordinary black coloration of the actinomycotic granules at a post-mortem examination, mentioned by Sir Holburt Waring, might be due to iron collected by the fungus in addition to calcium.

JOINT DISCUSSION No. 6.

Section of Psychiatry and Section of Neurology.

[*February* 11, 1930.]

DISCUSSION ON THE DIAGNOSIS AND TREATMENT OF THE MILDER FORMS OF THE MANIC-DEPRESSIVE PSYCHOSIS.

Sir E. Farquhar Buzzard : Some of us believe that cases of the manic-depressive psychosis, milder than those met with in mental hospitals, are frequently encountered by all who are engaged in the practice of medicine. If this belief is well founded, it is a matter of some concern that the fact is not emphasized in text-books. If the diagnosis of mild cases is often difficult, and their care a matter for skill and understanding, the absence of guidance for practitioners must be regarded as remarkable, if not deplorable.

While it is true that the clinical picture of this psychosis, with the high lights and deep shadows familiar to the psychiatrist, is often adequately described in text-books, chapters on neurasthenia contain no evidence that the features which distinguish neurotic from psychotic forms of depression have received due recognition. In the motley of neurasthenic symptoms, those with a manic-depressive taint are given no characteristic colour, and it is one of our duties to determine how far this distinctive coloration is possible.

We shall probably agree that there are mild forms of the psychosis which differ only in degree from the severe forms, and do not therefore present great diagnostic difficulties. The definite alternation of elation and depression and the periodicity of attacks are sufficient for labelling purposes.

But we frequently see depressed patients who do not give this history of preceding elation or depression. A source of anxiety may be ascertained and its importance as an ætiological factor has to be measured. The sequence of events suggests that anxiety precipitated or caused depression, but further inquiries are necessary. Is the anxiety still operative? If not, has its disappearance been attended by improvement? What has been the patient's customary reaction to anxieties of similar or greater gravity?

Although no definite story of preceding elation has been volunteered, inquiry may elicit a tale of restless activity or of unusual aggressiveness in the affairs of daily life. How does the patient describe his depression? A cloud that never lifts and never can be lifted; not exactly a depression but a detachment from reality; a loss of interest in everything and everybody and a loss of natural affection, for all of which he blames himself. Had he behaved otherwise, this would not have fallen on him, and he is not disposed to attribute his troubles to the faults of others. Am I right in thinking that the neurotic throws the responsibility for his troubles on others while the psychotic is ready to shoulder it himself?

While the depression covers his whole outlook, it is frequently centred on some event which hardly justifies his gloom. This concentration on one particular cause for his misery is not always present and its absence cannot be regarded as contra-indicating the diagnosis of a psychotic depression.

The depression, again, is always at its worst in the early morning and is little influenced by environment. Mental or physical fatigue will add to its severity. It may lift for a few hours at a time, without apparent reason, especially towards the end of an attack.

Fears are often associated with the depression—fears of insanity, of cancer, or of syphilis; or there is only a vague and objectless apprehension.

The power to grieve generally fails and tears are not easily provoked by emotional appeals. But some patients may weep copiously without being able to assign a cause, beyond their feeling of misery.

The patient's insight is usually well preserved but he complains that he cannot concentrate and cannot make a decision. His apparent defect of memory is the result of failure in attention.

Insomnia is not so invariable, or so difficult to overcome, as it is in anxiety states. I regard the absence of insomnia in a depressed person as pointing to the psychotic rather than the neurotic diagnosis. On the other hand, so great is the dread of the depression on waking that some patients fear to sleep and will take steps to avoid or postpone it.

The psychotic has more to say about his brain than about his other organs, and will describe periods of uncontrollable and incoördinate activity—"brain-racing"—as well as periods of mental stagnation, inertia and confusion. The prospect of recovery is inconceivable, and distraction from his distressing thoughts is only temporarily, if ever, obtainable. Morbid thinking, especially in relation to self-destruction, is an almost constant feature when sought for, and it is often difficult to determine whether attempts at suicide are impulsive or long premeditated. In most cases the latter is the more likely.

The neurotic is more concerned with his heart or his bowels, distraction is more easily achieved, and though he may express the desire to end his misery by death he is very unlikely to take the steps necessary to encompass it. The possibility of recovery is not only entertained but acknowledged, though the responsibility for successful or unsuccessful treatment is thrown upon others.

Psychotic depression is always associated with physical disturbances. There is usually an alteration in appearance, due to the combined effect of a number of slight changes. The eyes are dull, the hair loses its natural lustre and wave, the skin becomes sallow and pigmented, there is lassitude, loss of appetite, constipation, and a furred tongue. Indeed, few of these patients in these days can hope to escape the diagnosis of intestinal toxæmia!

I am not sure that the normal personality of individuals subject to these attacks is sufficiently constant to be of great value in diagnosis. They are usually well-endowed in regard to intellect, energy and conscientiousness, but may be lacking in confidence and initiative. Their diffidence and shyness may be well concealed by acquired mannerisms, so that they have a reputation for brightness and sociability, perhaps tainted by moodiness. When a woman of this description becomes less modest and retiring, more aggressive, restless and domineering, and this phase is followed by one of depression, the story is pathognomonic. But there are many cases in which no such history can be obtained, and some in which it is difficult to say when the real—normal—personality is present or absent.

More important than the patient's personality is his heredity. A history of mental disorder in the family has been assessed as high as 70% in the manic-depressive psychosis, and I lay stress on the frequency of suicides and of alcoholism. The former suggests that the manic-depressive predisposition is directly transmitted, and the latter confirms my view that alcoholism is often the direct result of this periodic psychosis.

Having referred to the difficulty of diagnosis in the mild forms—and the milder the form, the more difficult the diagnosis—and having indicated very briefly a number of clinical points which appear to me to be of importance in arriving at a conclusion, let me emphasize those which I have come to regard as most helpful.

(1) The type of depression, as described by the patient, and its chronological incidence and variations. (2) The loss of all natural and accustomed interests—in other words, the failure of the affective side. (3) The self-reproach and assumption of responsibility. (4) The preservation of sleep or, in many cases, the mild degree of

insomnia. (5) The history of a hypomanic phase, however vague, or of a previous attack of depression similar in character, but often of very short duration. (6) The coincident physical disturbances already described. (7) The family history, particularly of suicide and alcoholism.

In many instances a correct diagnosis can only be reached after much time has been spent in eliciting and elaborating the patient's account of, and attitude towards, his symptoms, and in carefully assessing the value of each. Even then one is sometimes left in doubt.

Treatment.—While admitting that there is a right way and a wrong way of treating patients suffering from the manic-depressive psychosis, and that the recognition of this principle is a matter of paramount importance, I have grave doubts whether we can rely on any therapeutic measure to prevent or shorten an attack. Let us consider prophylaxis first. A patient has recovered from a mild attack of depression which was attributed to overwork, and we are asked to advise in regard to the future. We feel compelled to warn him against excessive exertion, mental or physical, and to advise him to be temperate in all his habits. But while doing so we cannot forget the women, leading quiet, blameless and sheltered lives, on whom these attacks of depression have descended from time to time without apparent reason. Nor can we ignore the fact that if a hypomanic phase develops, our advice will be unheeded. Further, there will come to mind patients with the manic-depressive taint who have battled through times of prolonged stress and anxiety with complete success, only to break down when they are in smooth water. It is so easy to find a causal factor, so difficult to prove its guilt! The same scepticism undermines my faith in the efficacy of any treatment to shorten the attack. We know the patients who sink into depression every autumn and are cured by a new agency every spring. It was Coué one year, vaccines the next, and then hormotones, and we know that each year will add to the list. We have tried everything we can think of ourselves and, having given them up all in despair, our patients have recovered. And so we are reduced to the belief that the great majority of these patients will get well if we keep them alive, feed them, secure their sleep and guard them from the well-meant but ignorant efforts of their friends to rouse them from their depression.

It is difficult at times to decide whether a patient should be allowed to continue at work, and every case has to be decided on its own merits. If the patient's misery is obviously increased by his knowledge or belief that he is not efficient and that others may suffer in consequence, it is generally wise to insist on complete rest, the medical attendant taking entire responsibility for the decision. On the other hand, there are occasional circumstances in which the patient may, with advantage, be allowed to carry on his occupation. The danger of suicide must never be overlooked; for this reason, congenial and tactful companionship is a necessity, sleep and sufficient food must be secured, and disorders of digestion should be corrected. If there is any further therapeutic measure which can be relied upon to shorten an attack of depression, I shall be glad to hear of it, though perhaps slow to believe in it.

But when all is said and done, there is no disorder of mind or body in which the attendance of an understanding doctor is so necessary to his patient, not only as a guide and comforter, but as a safeguard against the stimulating and ignorant attentions of friends and relatives.

Dr. H. Crichton Miller: Let me say at the outset that I regret that the title of this discussion should have been worded as it is. The term "manic-depressive psychosis" may be correct enough for use in mental hospitals, but it suggests too much. The term "cyclothymia," on the other hand, covers the subject under discussion: it includes all the milder manifestations, and brings the whole syndrome within the frontiers of everyday life. It is important that the profession should

realize how widespread is the tendency to cyclothymia, and as long as we use so forbidding a term as "manic depression" that realization will be slow to come.

The complete clinical picture of manic depression is easily recognized when an accurate history is available. But the partial or early picture is much more elusive. Many physicians seem to regard as cyclothymics patients who are merely unstable. I suggest that the basic feature of cyclothymia or manic depression is variability of affective response, independent of the nature of the stimulus. The variability of the cyclothymic is instability only in the sense that a pendulum is unstable. He is not variable in the way that a boat with insufficient keel is unstable. Yet, if we allow ourselves to be misled by the histories given by relatives and sometimes by patients themselves, we shall conclude that for each change of phase from normality to exaltation, or from exaltation to depression, there has been an external cause. The true cyclothymic varies for endogenous reasons, and all other explanations are fallacies of lay observation. On the other hand, the large class of temperamental unstables do vary in response to emotional experiences which are too powerful for their capacity of adaptation. Thus the hysteric is often described as being either very exalted or very depressed, but rarely normal in her reactions. In a case of anxiety the patient may be described as fairly well adjusted until some situation of apprehension throws him into profound gloom. In such cases the relation of the stimulus to the response is all important for diagnostic and therapeutic purposes. But with the cyclothymic it is otherwise. When he is exalted, a trivial stimulus of a pleasurable order produces an exaggerated affective response; when he is depressed, painful stimuli are opportunities for expressing the misery he feels. In other words, the contrast between the various groups of temperamental unstables on the one hand and the class of cyclothymics on the other, lies in the difference of personal integration. The unstables may be regarded as badly integrated personalities; the cyclothymics present two contrasted integrations, each in its way imperfect. At any rate we may say of many cyclothymics, even in the advanced stages, that during phases of normality they present every appearance of a well integrated personality, whereas of the temperamental unstables this can never be said. We may, perhaps, formulate this idea as follows: the cyclothymic is an individual in whom the response to stimulus, both in feeling and expression, ceases during given phases to be appropriate to the values of the normally integrated personality.

It is therefore evident that to study the milder examples of cyclothymia, we must be acquainted with the patient's normal personality. This may be true of all psychopathology, but in no direction is it more important than in this. The exalted and depressed phases must be studied in relation to an actual or hypothetical norm. This is in contrast to dual personality, where, theoretically at least, one of the two contrasted phases constitutes a true integration of the personality. But the differences between cyclothymia and dual personality go deeper. In the latter there is dissociation, which need never be present in cyclothymia. The difference between Dr. Jekyll and Mr. Hyde was not a mere change in affective values, but one that depended on discontinuity of personality. In the true cyclothymic the difference of response depends not on associative, but on affective, variation. Indeed one of the most striking features of manic depression is the degree of maladjustment that can be reached before any rational failure manifests itself.

It is tempting to compare the two phases of a cyclothymic with two normal types—to say, for instance, that in his exalted phase the cyclothymic approximates to James' tough type and in his depressed phase to the tender. Perhaps it is more tempting still to say that extraversion, in Jung's sense, characterizes the one phase and introversion the other. But this is only a partial truth. To make it adequate we would need to say that in the exalted phase the cyclothymic is an extravert with the functions of thinking or sensation activated, while in his depressed phase he is

an introvert with exaggerated feeling or intuition. But even so we are left with an unconvincing picture, because the essential depression is inability to experience feeling that is appropriate to the stimulus. And in a sense this is the hall mark of his exaltation too, for in that phase he is the tough extravert, responding boisterously to many stimuli but perhaps failing in appropriateness of response as much as in his depressed phase.

Kretschmer, in his attempts to simplify type psychology, has made the fundamental contrast between cycloids and schizoids. This seems unfortunate. The introverted schizothyme has for his true contrast the extraverted hysteric. It would be approximately true to represent the cyclothymic as one who swings from the introversion of the schizoid to the extraversion of the hysteric. The reduced responsivity of the schizoid is shared by the depressed cyclothymic, just as the exaggerated responsivity of the hysteric is shared by the exalted cyclothymic.

If we seek an explanation of the periodic variation in reaction of the cyclothymic, we meet with nothing but negations. On the psychological side we find that no theory fits and no treatment is successful. It is of course true that many cyclothymics pass from depression to normality during, or immediately after, some form of psychotherapy. It is also true that a cyclothymic treated by mental analysis during his phase of normality may profit appreciably by emotional release and become thereby better able to carry on his life within the limits of his normal phase. But the cures of manic depressives by psychotherapy as by any other form of therapy tend to be fallacious. The *propter hoc* cure shows itself to be a *post hoc* cure when it is tried a second time. Thus the cyclothymic in his earlier phases of depression may attribute a " cure " to a holiday, to electrotherapy, to a sea voyage, to injections, to a surgical operation, or to Christian Science. But when the next depressive phase occurs the same treatment is almost certain to fail, unless it is resorted to at the time when he would in any case tend to emerge from his gloom. And when the cyclothymic passes from depression to exultation with no intermediate period of stability, he is apt to be a very enthusiastic propagandist and to proclaim the efficacy of the alleged cure with an undiscriminating zeal which, in the light of future disappointment, is liable to be regretted. Even palliative medicinal treatment which is appreciated while it is administered has no effect on the duration of the phase. Thus a depressed cyclothymic may admit temporary benefit from some tonic treatment and even ask that it should be continued. And the relatives of an exalted patient may appreciate the value of some sedative treatment which makes him more tolerable in the house. For my own part, in only three cases have I entertained any real hope, and in all three the hope has been shattered. In all three the rhythm was a fairly exact one. The patients had been subject to cyclothymia for periods of from ten to thirty years. In one case an annual rhythm was obliterated for two and a half years, apparently by the administration of ovarian extract. In another a long rhythm of four years was extended to eight, and the only explanation available was the treatment of a serious and long-standing dental abscess. In the third a rhythm of less than a year was replaced by a period of four and a half years' normality as a result of a satisfactory marriage. All these cases have since relapsed. I am very ready to believe that in the first two there was no causal connection between the alleged cause and the improvement. In the third, however, I am still inclined to believe that marriage and increased stability were causally related.

But in general we may say that all the researches made by psychiatrists and pathologists leave us with Kraepelin's " great psychological riddle " still unsolved. No one has succeeded, so far as I am aware, in establishing any correlation between physiological changes and the affective variations of cyclothymia. It is not a change from sympathicotonia to vagotonia; it is not dependent on an altered blood-sugar ratio; the basal metabolic rate remains constant; and so on—one might catalogue a long list of investigations that have failed to throw light on the essential character of the changes.

Nevertheless, certain fixed points remain to serve the purpose of finger-posts for our speculations.

In the first place, periodicity appears to be a physiological rather than a psychological quality. It is, therefore, reasonable to look for a somatic rather than a mental explanation.

In the second place, there is a great similarity between the euphoria of alcoholic intoxication and the exaltation of the cyclothymic. This suggests that chemical action, presumably on the thalamus, is the basic factor at work.

Thirdly, there is a good deal of similarity between the type of depression associated with chronic intestinal absorption and the depressed phases of cyclothymia. This suggests again an endogenous chemical factor.

Fourthly, the commonest example of cyclothymia occurs in some women in relation to the menstrual cycle. We can, indeed, see the most exact reproduction on a small scale, of exaltation, normality and depression within a period of twenty-eight days. Furthermore, it is admitted that of all manic depressives a relatively small proportion are leading a physiological sex life. This suggests that the biochemical factor may be hormonic.

We might, therefore, narrow down our pathological speculations to something of this nature : A rhythmic disequilibrium of hormonic function which tends to be self-limited, which results in an endogenous toxæmia, affecting thalamic activity in two contrasted ways, and influencing cortical activity only in a secondary manner.

The problem is not one for the psychologist, but for the biochemist, and its solution will only be discovered within the complex metabolism of the organism in its chemical response to functional activity.

Dr. George Riddoch : Stress has rightly been laid by the previous speakers on the apparently causeless onset of depression in manic-depressive psychosis. Whilst this is common, it is not invariable, for frequently emotional disturbances precedes attacks, and appear to precipitate them. Continuity of the depressive phase is of diagnostic importance. The neurasthenic has periods which often appear almost fortuitously, in which he feels "almost himself." These are more common in the afternoon, and with the psychotic it may be so too; but in his case the depression only "lifts" to some extent, as it does after a good night's sleep, but it never disappears except during convalescence. That the depressed neurotic cries easily, and so obtains some relief, whereas the depressed psychotic does not, although he feel that if he could he would be better, is of some diagnostic significance ; but again the rule is not absolute. The person who is depressed as the result of manic-depressive psychosis, however, differs more from the neurotic in that he is more apt consistently to believe that his illness is his own fault, and tries less to project the blame on to others and external influences. His depression is little diminished, even temporarily, by reassurance.

The mental and physical sluggishness of the individual with psychotic depression is of great importance in diagnosis. Difficulty, as well as slowness in thinking, the sad expression, general immobility, as well as the poor appetite, sluggish bowel, sexual apathy and cold extremities are significant. Hallucinations in the manic-depressive, even in mild attacks of depression, are not uncommon, although they may be evident in only one attack.

From the point of view of the general physician, one must remember that the manic-depressive patient, in a state of depression, may come complaining of anything but his depression. The symptoms which he first describes may, for example, be headache, abdominal discomfort, constipation, sleeplessness, or pain in the chest, and the underlying depression may only be admitted after some time, and then with reluctance. It is kept back, perhaps, because the associated suicidal ideas have become intense.

A history of previous attacks of depression, with or without intervening periods of elation, especially if they are seemingly causeless, is important, but the second attack may develop long after the first, as in a case in which there was an interval of twenty-five years between the attacks, both of which were precipitated by anxiety. Inquiry into the family history should never be omitted.

Treatment.—There is no known cure for this form of illness, which is due in part to a constitutional defect. We are not, however, completely helpless in regard to it. Something can be done to diminish the patient's misery by placing him in a suitable environment and giving him sleep, if necessary by the use of hypnotics. Such patients are usually better dealt with away from home, but a mental hospital is not the best environment in the milder states of depression. They may seek admission there as a protection from suicide, but they are often acutely aware of their surroundings, and are, on recovery, unhappy in the memory of them. A home in the country, like the Cassel Hospital, endowed, and with an adequate nursing staff, would be a great boon, and would be most suitable for the care of patients suffering from recurrent depression.

The importance of tact and patience on the part of doctors and nurses cannot be over-estimated. Psycho-analysis is to be condemned, as it leads to aggravation of the symptoms.

The danger of suicide has to be kept in mind, even when the depression is slight. The danger is particularly great during convalescence, when the patient may simulate cheerfulness in order to be discharged and so have a chance to end his misery.

Dr. Henry Yellowlees said that the first and most important point which this discussion presented was the differential diagnosis between neurasthenia and psychotic depression. The view existed that there was only a difference of degree between the patient who was feeling a trifle downhearted and the patient who was psychotically depressed. Against that idea he (the speaker) strongly protested. The popular phrase that melancholia was a caricature of normal depression was misleading, unless it was made clear that caricature implied a difference in kind as well as in degree. For the same reason he looked with suspicion on the phrase "mild melancholia." Of course, the psychosis under discussion might show various grades of acuteness or intensity, but in stressing the term "mild" there was a tendency to forget the melancholia. It was not customary to speak of "mild pneumonia." Pneumonia was never a mild illness, though some attacks were less severe than others.

The diagnosis between neurasthenia and the form of melancholia with which it was often confused was a clinical problem, the crucial point being that the neurasthenic was a person of active emotional reactions, who bewailed the limitations which his illness imposed upon him; whereas the melancholic was the reverse. It was now the fashion to regard Kraepelin as somewhat old-fashioned, and no doubt much of his psychology and terminology were out of date; but that great authority knew much more about the problem under discussion than the majority of those who succeeded him. To make this diagnosis was perhaps the most responsible and most difficult task in psychological medicine, and without an understanding of Kraepelin's view-point it became a matter of more or less intelligent guess-work.

Two other important points to which he would allude were: a recognition of the cardinal neurasthenic symptom, namely, excessive fatigability, and the final and subtle touchstone, namely, the presence of insight in the neuroses and its absence in the psychoses. The psychotic, however mild his illness, had not insight. He (the speaker) had never known that touchstone to fail.

There was one essential for the physician in this matter: he must have an intimate knowledge of the natural history of the disease. All made errors in this

matter, but the man whose practical training in and acquaintance with the psychoses began and ended in his own consulting room, would make more mistakes than others would do.

Mild simple elation was undiagnosed more often than any psychosis which he (the speaker) knew. This was probably due to lack of training which blinded one to the wood because of the trees. One often heard the remark, "He is sane; he has no delusions." A few months ago a patient entered his consulting room, not as a patient, but "for a friendly chat," though he (Dr. Yellowlees) had never seen him before. The man told him of his income, his investments, and the provisions of his will; he informed him of his wife's Christian name, the circumstances of his proposal to her, and all the chief events of his honeymoon. He then went on to impart his views on politics and religion, and also on sexual morality. He then inquired after his (the speaker's) family and finances, and asked if he had any difficulty in securing a regular daily action of the bowels. He reassured him on the point, and the patient finally invited him to dine with him, and left saying he could rely upon him. Apart from the closing remark, Dr. Yellowlees had no reason to doubt the truth of anything he had said. He had no delusions, but he was desperately ill, he was psychotic, and was a hypomaniac.

The difficulty in looking at such cases as a whole was illustated in Courts of Law. The majority of the psychiatric *causes célèbres* hinged on this question of mild simple mania, and the procedure was always the same; physician after physician entered the witness-box, and a series of isolated acts of the patient were described, and in connexion with each one of them came the query, "Is that a sign of insanity?" He (Dr. Yellowlees) was hoping that someone would protest against the iteration of this meaningless question and insist upon describing the mental state of the person as a whole, and in his own way.

Surely there was some misapprehension of the meaning of the word "treatment." He took it that previous speakers would agree that there was a treatment for pneumonia, and a treatment for enteric fever; but it was agreed that whatever treatment was applied, one was unable materially to alter the date of the crisis in pneumonia, or the gradual fall of the temperature in enteric fever. But the fact that an illness had to run its course hardly justified one in saying there was no treatment for it. The treatment depended on the proper conception of manic-depressive psychosis as a whole. For many cases, especially in the manic phase, "management" was a better word, because it was true that in a sense mild mania was untreatable. His (the speaker's) point was concerned with the difference between melancholia and neurasthenia. Melancholia was a psychosis, and it was, *ipso facto*, not amenable to psychotherapy. Neurasthenia, on the other hand, was amenable to psychotherapy in general, and was often especially amenable to analysis in particular. The diagnosis of melancholia needed courage, and its treatment—a very real thing—needed time and patience, experience and specialized skill. Therefore it was not surprising that attempts to treat melancholia on psychological lines were frequently made, and his mind had been relieved to hear such weighty opinions as those of previous speakers expressed against such a proceeding. Psychotherapy, in one sense, was an important adjunct to treatment; it encouraged the patient and relieved his suffering by suggesting hope, etc., but he (Dr. Yellowlees) was referring to the word in the modern sense.

Psychopathology explained many of the mechanisms of the disorder. It explained why it was so much better to treat the patient out of his own house, and also explained many of the symptoms and mechanisms, in both the manic and the depressed phases of the psychosis. But there was always a wealth of material to be analysed in a depressed patient. Cure of melancholia by analysis was impossible. If a physician attempted such a thing, there were only two explanations; the charitable assumption was that he knew nothing about melancholia!

He (the speaker) was not happy about this question of the treatment of melancholia. One could not read the newspapers for a week without seeing a report, usually of an inquest, with the ominous line, "His doctor had been treating him for depression." Whatever efforts were made to explain it away, the danger of suicide in any form of manic-depressive psychosis was an ever-present one, and it was the duty of the physician to refrain from adding himself to the dubious company of those who treated patients for depression with the possibility of such a result as an inquest.

Dr. W. R. Reynell: The diagnosis of a fully-developed manic-depressive case presents no great difficulty, but it is otherwise with the mild cases. The first attack may consist of a single phase, usually depression, occurring several times before the complete cycle establishes itself. I am not convinced that the depression in these cases is always of the same kind. Some will have feelings of sin and remorse, others ideas of poverty, of disease, of inferiority, and of resentment against others, the character of the morbid ideas depending upon the patient's personality. Sooner or later the other half of the cycle will develop, with feelings of euphoria and reversal of affect. We cannot make any hard-and-fast classification of these mild types, which shade off into anxiety cases and minor forms of emotional instability, on the one hand, and into the graver psychoses on the other. We cannot distinguish sharply between psychoneuroses and psychoses. The difference is one of degree rather than of kind. Complex entities cannot be classified into "either" . . . "or," they are usually both and sometimes neither! A "depressed" case is not always a purely neurasthenic or a purely manic-depressive case—it is often both and sometimes neither, e.g., a schizophrenic. Most cases in fact are mixed, and "borderland cases" sometimes show features of psychoneuroses and, at other times, of neuroses or psychoses. Manic-depressive cases are often abnormally fatigable, like neurasthenics. They may have fainting fits which are on the borderland of epilepsy, and in rare cases true epileptic fits occur. Migraine occasionally undergoes what has been called the "epileptic transformation," especially at the menopause, and in some subjects the attacks are replaced by sudden fits of depression without headache and often preceded by a short period of euphoria. Epileptic fits may be replaced by "psychic equivalents," and even the depression of an attack of gout may be preceded by feelings of euphoria. All the periodic psychoses and neuroses seem to be related, both in genealogy and in manifestations. More than 80% of "nervous" cases show a "nervous" heredity often including both psychoneuroses and psychoses. Surely all this points to some inherited somatic defect. "Psychotic potential" is perhaps as good a name as any. If there is such a factor it will vary from zero to the maximum, in any large number of persons. We may suppose that if the inherited defect or psychotic potential is low, no amount of conflict, mental stress, or toxæmia, will produce psychotic symptoms, but may be enough to produce psychoneuroses. If the psychotic potential is higher, psychoses may develop with exceptional conflict, stress or toxæmia, and in patients with a still higher potential, psychoses will be caused by comparatively slight psychogenic or exogenous factors.

Again, let us consider a large number of "nerve" cases with neurotic inheritance. We would expect that on the above hypothesis, if we plotted the number of cases against the psychotic potential, estimated by frequency of psychoses in family history and by other means, we would get a "normal" curve of distribution. If a patient has an inherited tendency to develop any periodic affective psychosis such as a manic-depressive cycle, or indeed such symptoms as headache, migraine, epilepsy, dipsomania, asthma, or insomnia—the actual attack may be precipitated by endogenous or exogenous causes, by internal or external disharmonies, by psychogenic or physiogenic factors. The "trigger" is in fact psycho-biological. The patient with low nervous potential may suffer from any or all of the common nervous symptoms

—migraine, insomnia, fainting fits, depression, anxiety, indecision—and the rest. The neurasthenic often shows superimposed cycles of euphoria and depression, and may or may not develop into a typical manic-depressive case. My views on treatment follow naturally on what I have just said. The greater the psychogenic factors the more psychotherapy is indicated; the greater the somatic factors, the more we will rely on rest, isolation and physiotherapy. The particular kind of psychotherapy that is needed will depend upon the individual case—in some "scratching the surface" will suffice, in others the surface must be "ploughed up," and in a few "deep mining" may be necessary.

Dr. Helen Boyle: I wish to deal with the connection between mild manic-depressive, or cyclothymic tendencies, and endocrine—more especially thyroid gland—disturbance, i.e., hyperthyroidism, hypothyroidism, and possibly dysthyroidism. This relationship, if true, may have some bearing upon both the diagnosis and treatment of these conditions. My cases, some of them under observation more or less for twenty years, have been mainly of this mild uncertifiable cyclothymic order. In some of them attacks of insanity have developed, and they have passed on to mental hospitals, in others the patients have died.

From observations in hospital and private practice it seems to me that there is too high a percentage of manic-depressives who show evidence of thyroid disturbance for the connection to be due to coincidence. Out of the last hundred private cases thirteen were manic-depressive or cyclothymics, and in six of these there had been thyroid trouble at some time, others had a rapid heart and other suggestive evidence, while of the other eighty-seven only one (a schizophrenic) had a large thyroid. Out of 100 consecutive cases at the Lady Chichester Hospital, six were cyclothymics, and in four of these the patients had large thyroids. Of the ninety-four others only eight had large thyroids.

In some manic-depressive cases there is a history of definite enlargement of the thyroid during adolescence, and evidence of mental or nervous instability of a cyclothymic type accompanies the thyroid enlargement when it appears. There seems to be no essential difference, except in degree, between the mild non-certifiable cases, and those in which the patients become so much excited that they must be regarded as manic, or so much depressed that they are suicidal. Kraepelin's classification definitely includes these mild cases, and he observes that thyroid enlargement is sometimes present.

Dr. Golla, in his interesting paper, "The Mental Symptoms in Hyper- and Hypo-thyroidism," said:—

> "We should expect that the cardinal feature of the mental disturbance accompanying hyperthyroidism would be of the nature of a general hypermotivity complicated to a greater or less degree by the exhaustion æsthenia following on the excessive metabolism."

Later, he says:—

> "Exacerbations occur in which the mental symptoms resemble in many ways those of manic-depressive insanity, from which they may to some extent be distinguished by their greater mutability and the ease with which they can be influenced by external suggestion."

The qualification "to some extent" shows up the difficulty, and possibly the lack of suggestibility is due to the severity of the disease rather than to inherent differences. I submit that some mild manic depressives are open to suggestion.

Again, he says:—

> "Symptoms of hyperthyroidism are not infrequent in many well-developed cases of manic depressive insanity and agitated melancholia and in such cases periods of excitement correspond with the appearance of symptoms of hyperthyroidism and subside when the thyroid symptoms abate."

When we compare the description of the mental and physical states in mania and melancholia with those in hyper- and hypo-thyroidism, it is interesting to see how much they have in common.

Manic-depressive disturbance is commoner in women than men, so is Graves' disease, estimated as high as ten in woman to one in man, and in myxœdema seven women to one man. The usual age for the onset of Graves' disease is from 16 to 40. Manic-depressive insanity also usually begins between these ages.

A point which is of interest in view of the possible connection with the thyroid is the immunity from ordinary infection—such as "colds," influenza, etc.—which some of these patients appear to possess during some phases of their illness. One said: "I never get a cold when I am ill like this." The immunity is only temporary, for when they are in their normal phase they are once more vulnerable, a fact which appears to indicate something not purely psychogenic in the disease, or at least a physical result of the disease.

In one case of Graves' disease, of which I had the care six times from 1916 to 1928, when the patient died, there was marked alteration from hyper- to hypo-thyroidism. The patient had a large thyroid, which varied considerably in size, and she showed cyclothymic tendencies. She had typical excited attacks, with flight of ideas, desire for unwise enterprise, letters fifteen pages long, motor excitement, unfatiguability, etc., followed by intense depression, even to the extent of suicidal attempts.

From the psychogenic side it is stated that the conflict at the root of this disorder dates from the third or fourth year, and is a conflict between the ego and the super-ego (Stoddart). But apart from this, fear is a potent source of thyroid trouble and it may well be that fear of some aspect of life is at least a contributory source of the thyroid enlargement at adolescence, even such superficial and obvious fears as fear of failure in examinations, or in games, or in the gymnasium, or in popularity. Is it possible that this fear of failure, based on deeper conflicts, and the intensive striving to attain, stimulated by the thyroid secretion, may be succeeded by exhaustion, thus establishing a cyclothymic tendency?

There are many and various indications of the parallel appearances in cyclothymia and in states due to disturbance of the thyroid. Kraepelin remarks that, while in the manic attacks the scanty hair grows afresh, in the depressive phrase the skin is dry, rough, the eye dull, the growth of nail stops or is irregular, and the menses are scanty or intermittent, a state which suggests hypothyroidism. In Price's "Textbook of the Practice of Medicine" the onset is stated to be, as a rule, between the ages of 20 and 30, and the onset of Graves' disease between the ages of 15 and 30.

The incidence, as regards sex, is stated in Price's "General Medicine" to be 6 women to 1 man in Graves' disease cases, and of certified manic depressives in Kraepelin's clinic, 70% are women—a large preponderance of women. There are no figures for the milder uncertifiable cases.

The functional activity of the normal thyroid differs at different times of the year. This also applies to many manic depressive cases which show annual variation.

Again, in Price's "Textbook" it is stated that in Graves' disease, the origin of the thyroid trouble is possibly due to stimulation of the cervical sympathetic by suprarenal gland extract or alimentary tract toxins. If the former, it is of emotional origin, if the latter, it is due to an infection, but evidence is inconclusive. One observer found 72% had a history of psychic insult and local infection in 40% cases.

It is said by some that manic depressive insanity is a "pure psychosis," therefore possibly emotional in origin.

In Graves' disease carbohydrate oxidation is said to be modified to cause glycosuria.

Kraepelin quotes that in manic depressives alimentary glycosuria occurs in 67% in depression, in 19% in mania.

Treatment.—A few cases seem to be helped in the depressed state by endocrine medication, but in those in which there is much excitement in the manic phase this treatment appears to be of no use in the succeeding asthenic state, the whole organism is played out. The condition of the pulse is a help here and so is regular weighing. In the still milder cases one of the chief needs is employment well within the powers of the patients and where they will obtain appreciation and encouragement, where no severe effort is ever needed, and where there is no power to disorganize the work by optimistic and ill-balanced schemes in the euphoric stage. The patients should in the normal stage be informed, so far as it is possible without depressing them, of the probabilities of the disease. The knowledge helps to steady them in the manic phase and to re-assure them in the depressed phases.

They should keep in touch with a doctor whom they can trust, as severe attacks may supervene. If it is true that these cyclothymics begin in many cases during adolescence, and that enlargement of the thyroid is associated with the disease, it follows that these adolescent cases should be guarded from all strain, anxiety and competition and that they should be watched for.

In one case deep X-ray therapy was used, but though it seemed to steady the pulse and nervous condition, the patient died not long afterwards from some ill-defined internal mischief, with severe diarrhœa.

Apart from symptomatic treatment, medicines appear to be of little avail, but they are of great value in relieving symptoms. Rest in bed is the most useful treatment, especially with plenty of fresh air, and massage when required.

In conclusion, I would enter a plea for the whole group of manic-depressive disorders to be reconsidered, with a view to possible subdivisions.

Dr. Neill Hobhouse: Sir Farquhar Buzzard has made it clear that the recognition of these cases by the profession in general has not yet been achieved. I think that in the differential diagnosis of the manic-depressive psychosis in the early stage the question which arises most frequently is just the one which he has discussed so fully, i.e., the diagnosis between this disorder and an anxiety state. I recognize that the conditions to which I shall refer are not such common sources of difficulty, but my excuse for mentioning them is that they are for this reason more likely to escape notice.

After one has come to the conclusion that a patient is suffering from depression which is endogenous, fairly abrupt in origin, and out of keeping with his previous mental habit, there remains the question whether one is dealing with a case of manic-depressive psychosis or something else. There are one or two conditions which produce a similar mental state, but should not cause much difficulty. An early stage of general paralysis of the insane may do this, and may simulate either an exalted or a depressed type of cyclothymia. Another disorder is myxœdema; it should not present great difficulty in diagnosis, and I would hardly have mentioned it if I had not seen, during the last year, a woman who had been certified and retained in a mental hospital and who was suffering from a severe myxœdema which had not been treated. If an attempt is made to study depression as a symptom, I think that hypothyroidism certainly deserves its place among the causal factors. There is another condition which deserves a prominent place in the differential diagnosis of depressive states, the milder manifestations of meningo-vascular syphilis.

Some years ago I examined a number of ex-Service men who had developed nervous symptoms and made a claim on the Ministry of Pensions. A great number were suffering from anxiety-states, frequently of a type closely allied to the litigation-neurosis. But there were a considerable number with mild depression, loss of

interest, mental retardation, without confusion but with occasional bursts of temper, whose past history and general outlook on life were very unlike those of the psychoneurotic. I was struck by the number who had been treated for syphilis. I investigated most of these cases, and it was not rare to find a positive Wassermann reaction and a moderate increase in cells in the cerebrospinal fluid; the blood was less often positive. The cerebrospinal fluid and the response to anti-syphilitic treatment made it clear that they were not cases of general paralysis of the insane. Under anti-syphilitic treatment nearly all made great improvement, losing their depression, but not always regaining their former efficiency. I have seen several cases more recently, in which a history of depression typical of that which we are discussing, has been given by a patient who had signs in the cranial nerves of meningo-vascular syphilis, and I believe that this disorder may produce a mental state which can simulate that of manic-depression. I stress particularly the cases which had received treatment in the past, for it is common for a patient to have treatment for syphilis, and to think no more about it. I take it that in these cases the treatment had warded off the more serious symptoms—the confusion and intellectual impairment, as well as the cranial nerve palsies—but that a recrudescence of disease had produced the change of affective state. The response to treatment showed that they were not due to residual vascular changes. My experience has led me to regard meningo-vascular syphilis as a possibility to be considered whenever one meets with a case of endogenous depression of recent origin with no history of previous attacks.

The phase in which there is an acceleration of mental activities and speech, and a raising of the affective state, is not so common or so serious as the depressive variety, but in a mild stage it often comes the way of the neurologist. These patients will probably be clear of the dumping ground of neurasthenia, but there is a lesser dumping ground of hysteria in which they are often deposited. The features which they have in common with hysteria are excitability, and the rather noisy combativeness with which they respond to any opposition. Fundamentally they are different from the hysterics; they do not feel ill, do not pity themselves or desire the pity of others, and their behaviour is not actuated by desire to attract attention. Nearly always they have come to the doctor under pressure, because they have become difficult to live or work with. The high value which their euphoria gives to their own opinion is frequently the factor which gets them into trouble; they are unable to refrain from impressing their views on their superiors, and from suggesting improvements in their neighbours' mode of life. In my experience they have mostly been women between the ages of 25 and 35, and have been of a definitely efficient type, unlike the usual subjects of hysteria. I expect that most of us have come across cases of hyperthyroidism in which the psychic changes seem to have preceded the physical ones; in which the tremor and tachycardia were not more than might be met with in a somewhat excited patient, and in which the customary fatigability and malaise were masked by euphoria. I have often seen similar cases in which there *was* some enlargement of the thyroid, and I have more than once been in doubt when there was not. The B.M.R. is not as helpful as might be expected, as it will probably be on the border of the normal. One thing which emerges from this discussion is the general agreement that the clinical entity which we are considering requires greater recognition by the profession. If these cases are to be recognized they must be recognized not as "mental cases," but as patients who turn up in out-patient clinics and in practice. I should like to discard the term "manic-depressive psychosis." I am sure that it is important that a new name should be adopted; I think it would be advantageous if this name sounded more distinctive, more suggestive of a clear-cut entity, than an expression such as endogenous depression. The word "cyclothymia" seems to suit the purpose as well as any. Might not its adoption in textbooks mark a step in the fulfilment of the object which inspired this discussion?

Dr. E. B. Strauss: For several months past I have been an assistant in Professor Kretschmer's Psychiatric Clinic at the University of Marburg, and my views on the subject under discussion are therefore largely those of the Kretschmer School—a school in direct succession to those of Kraepelin and Bleuler.

I shall restrict my remarks to the depressive side of manic-depressive psychosis. During our first years of clinical psychiatry, we are struck by the extreme frequency of manic-depressive psychosis; later, this impression is corrected, and we are forced to the conclusion that this psychosis is much more uncommon than we previously thought. The implication is obvious, namely, that the diagnosis was too comprehensive and that our conceptions of manic-depressive psychosis as a clinical entity have undergone considerable modification in the light of the work of modern psychiatrists, especially that of Kretschmer. By no means all depressive attacks, even periodic depressive attacks, should be diagnosed as manic-depressive psychosis. Differential diagnosis is of paramount importance, for on that depend both treatment and prognosis. Depression may occur as the most prominent symptom in the manic-depressive psychoses, reactive depression, depressive catatonia, arteriosclerosis, dementia paralytica and senile dementia; and it may largely colour psychoses of known toxi-metabolic or dysendocrine origin. Manic-depressive psychosis is the second most important of the group of the endogenous psychoses. It is an unalterable congenital component of the patient's entire being. It is so much an entity, that it is intimately bound up with questions of heredity, and there are, corresponding to the psychosis, psychopathic temperamental characteristics (termed cycloid) and normal temperamental dispositions (termed cyclothymic), which are equally congenital and unchangeable. Further, there are definite affinities between circular psychosis, the cycloid and cyclothymic dispositions, and a certain type of physique, termed pycnic. Kretschmer does not maintain that a person of leptosomatic physique cannot suffer from true manic-depressive psychosis, or that pycnics are immune from schizophrenia; he merely emphasizes the striking affinities between the endogenous psychoses and certain physical types, and recent work has definitely proved that the course and symptomatology of manic-depressive psychosis and schizophrenia are greatly modified in the presence of unaffiliated physical characteristics, in the patient himself or his immediate family.

All this is of great importance in establishing the diagnosis of manic-depressive psychosis. The anamnesis is all-important and must contain a carefully taken psycho-biological record of the patient's immediate family, so that we can evaluate the schizoid-schizothyme, or cycloid-cyclothyme familial factors on the one hand and the predominant familial habitus on the other, and utilize this knowledge as diagnostic indices. The anamnesis must also contain a careful estimation of the patient's temperament and character in his periods of mental health. It is also important that the patient should be completely measured and his physical type established. Finally, the psycho-physical examination must include a search for traces of all schizophrenic stigmata; observations of the patient's motor idiosyncrasies is a great help in this respect. We are now in a position to make our diagnosis, give our prognosis, and prescribe the correct treatment. If we find that our patient has had previous attacks of depression, or is usually over-boisterous and genial, or has, in fact, a temperament and disposition which fits in with any of the cycloid or cyclothymic groups, and if we find further that his physique is predominantly pycnic and that pycnic and circular cycloid or cyclothymic traits predominate in his family, we can establish a diagnosis of manic-depressive psychosis with absolute certainty. We know that unless the picture is complicated by organic factors, such as arteriopathy or para- or meta-syphilis, the patient will recover, usually within six months; and we know the most appropriate method of treating the attack. To my mind, it is unthinkable that a person with a predominantly schizoid make-up could develop a typical attack of manic-depressive

psychosis. Depression occurring in such a subject should always arouse suspicion of a depressive attack of the catatonic form of schizophrenia, or of a mixed psychosis. In such cases the prognosis is always different and nearly always worse.

Just a word about reactive depression, probably one of the most common forms of depression met with in psychiatric practice, and a form most frequently mistaken for manic-depressive psychosis. This condition is precipitated by an intolerable situation in the patient's actual life. It is allied to true neurasthenia, prison-psychosis, and the like. Whether the condition is entirely exogenous or whether a current conflict stirs up and allies itself to unconscious psychisms, may be debated by psychoanalysts. It is certain that it is not endogenous and constitutional in the sense in which manic-depressive psychosis is so. Judging from my own experience, I should say that reactive depression is a disease of schizoid personalities (with or without the correlated physique) rather than of cycloid or circular personalities. Oddly enough, the cycloid faces real trouble with great composure and does not respond with a depressive attack. His depressive phases seem to bear no relation to the actual outside situation. It is in this reactive or situational type of depression that psychotherapy can be of most benefit.

Institutional treatment, to my mind, is desirable in even the mildest attacks of manic-depressive psychosis. In England there are three hospitals for nervous diseases and two or three special hospitals for uncertified mental cases. In Germany, every large town has its "Nervenklinik," which serves a whole district. In the Nervenklinik every type of nervous disorder, neurotic, psychoneurotic or psychotic is treated, and people think no more of being admitted into the Nervenklinik than into the "medizinische," "chirurgische," or any other kind of "Klinik." In England, the idea of admission to the Maudsley or Bethlem Hospitals, or as a voluntary patient in an ordinary mental hospital, is still terrible to the patient and his relatives. I believe that in an institution the mild cases do better with light occupational therapy than with complete rest. Agitated cases do well when there are no contra-indications (and there are many possible contra-indications) with continuous narcosis with avertin for about ten days. Completely inhibited depressives respond well to pyrexial treatment, which should be begun just as the patient emerges from his depressive apathy. In the case of women, whose depressive attack coincides with the climacteric, or occurs post-climacterically, a really active preparation of ovarian gland (I know only one such preparation) sometimes helps remarkably to shorten the attack.